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URETERAL STRICTURE—REPORT OF 100 CASES[1]

By Guy L. Hunner, M.D., F.A.C.S., Baltimore

(From the Gynecological Department of The Johns Hopkins University and Hospital)

Ureteral stricture or narrowing of the ureteral lumen due to intrinsic inflammatory changes in the ureteral wall is a disease far more common and of vastly greater importance than the literature or our previous experience has led us to believe.

In a preliminary paper read before the Genitourinary Section of the New York Academy of Medicine in January, 1916,[2] the author reported 50 cases of ureteral stricture occurring in his practice up to November 1, 1915. In the same period of 13 years of practice there were records of 49 cases of nephrectomy for tuberculosis and 39 cases of stone in the ureter. In the 18 months since November 1, 1915, or since becoming alive to the importance of the subject, and since looking for stricture, talking stricture, and having confrères refer suspected cases for diagnosis and treatment, the author has seen more than 100 additional cases.

This paper will deal in a statistical manner with the first 100 cases only, although the general conclusions will be based on an experience with over 150 cases, and a few of the illustrations will be of recent cases not in the first 100 but showing points of particular diagnostic interest.

Etiology.—Up to within the past few years most of the literature on this subject has been devoted to the so-called congenital stricture of the ureter.[3,4]

Kelly[5] anticipates our more modern literature of the subject by stating that "Strictures are caused by an inflammation in the ureteral walls produced by the commoner pyogenic cocci, by the gonococcus, and by the tubercle bacillus. The commonest form of inflammation is that due to the tubercle bacillus, and the rarest in my experience in women is due to the gonococcus."

Garceau,[6] on the other hand, says, after reviewing the literature, and in the light of his own experience, "The chief cause of fibrous stricture is gonorrheal infection."

Furniss[7] took issue with the prevailing opinion that most ureteral strictures are congenital in origin, and from a study of his cases concluded that infection plays the important part in the production of ureteral stricture. He thinks that the

[1] Paper read before The Johns Hopkins Hospital Medical Society, May 7, 1917.

[2] Stricture of the Ureter, Excluding Tuberculosis and Calculus; Report of Fifty Cases. New York Med. Jour., July 1, 1916.

[3] Bottomley: Certain Congenital Strictures of the Ureter. Annals of Surgery, 1910, LII, 597.

[4] Eisendrath: Congenital Stenosis of the Ureter. Surg., Gyn. & Obstet., 1911, XII, 533.

[5] Stricture of the Ureter. Jour. Amer. Med. Assn., 1902, XXXIX, 363.

[6] Ureteritis in the Female. Amer. Jour. Med. Sci., 1903, CXXV, 284.

[7] Jour. Amer. Med. Assn., 1912, LIX, 2051.

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infiltration in the ureter is the result of an acute hematogenous infection of the kidney, which often persists as a pyelitis, ureteritis, or secondary cystitis.

Furniss quotes Sugimura[8] as having studied the lower end of the ureters in the bodies of 25 patients, who had had cystitis but had died of other causes. He found changes of an inflammatory type in the submucosa and muscularis and was of the opinion that the infection extended through the lymphatics and not along the mucosa.

Necker[9] exhibited before the Deutsche Gesellschaft für Urologie, at the Congress in Vienna in September, 1911, pyelograms of cases of pyelitis, all showing some dilatation of the renal pelvis, and in explanation said that they were cases of ureteral obstruction with secondary infective hydronephrosis.

Kelly and Burnam,[10] speaking of traumatic stricture, voice our common experience in stating that "Traumatic stricture of the vesical end of the ureter following the injuries of labor and of surgical operations especially the Wertheim operation for cancer of the cervix uteri, is quite common. As a rule, the trauma has so interfered with the blood supply of the organ that there are lateral necrosis, a continuous leakage of the urine, and ureterovaginal fistula. The spontaneous healing of such a fistula, almost invariably, means stricture."

Gun-shot and stab-wound injuries are rare. Morris[11] collected reports on 5 such injuries. Figure 1 shows a stricture resulting from gun-shot injury and giving rise to symptoms 8 years after the injury.

Severe traumatic accidents are rarely the cause of stricture, as one would naturally conclude after consideration of the anatomy of this long flexible organ, well protected along the spine and attached at either end to a movable organ. Any accident severe enough to rupture the kidney or bladder or to crush the bony pelvis might well result in ureteral stricture, but the history would be such as to suggest the etiology in a case of this nature.

Syphilis should be kept in mind as a possible cause of ureteral stricture.[12]

Ureteritis cystica may cause ureteral obstruction from the large size of some of the cysts, but this condition, although probably of inflammatory origin, cannot be classified as ureteral stricture.[13]

Gynecologists might question the possible relationship between infected cervical glands and ureteral stricture. As we shall learn later, the vast majority of these strictures occur in the broad ligament region and it is only natural that we should suspect the cervix as having a possible causal relationship.

It is probable that ureteral stricture occurs more frequently in women than in men, and one is inclined to attribute this to the infections incident to childbirth. Opposed to this theory is the fact that in a considerable proportion of cases the symptoms of stricture begin in childhood or occur in women who have never married and in whom we have no reason to suspect a gonorrheal infection. Our failure to recognize the disease in both sexes has undoubtedly been due to our ignorance of its frequency and consequently to a failure to look for the lesion.

Although I have not seen any reason in any individual case for attributing ureteral stricture to a cervical infection, I have become suspicious of the etiological relationship between focal infections and certain cases of cervicitis. We have a type of urethral inflammation in which the vulva and paraurethral glands are inflamed and the patient has an inflamed cervix furnishing a considerable amount of leucorrhea. This type of inflammation is particularly likely to occur in adult maiden women, who show no other evidence and give no history of onanism or of gonorrheal infection. Time will demonstrate, I believe, that the group of inflammatory foci in the pelvis are secondary to a distant common focus of infection.

The frequent association of ureteral stricture and venous phleboliths in the immediate neighborhood speaks for a common origin for the inflammatory process resulting in the two conditions, and suggests that some of these infections may have originated in the genital tract.

While we must admit the possibility and probability of any of the foregoing factors playing a rôle in the etiology of certain ureteral strictures, I am firmly convinced that the majority of ureteral strictures, excluding those of tuberculous origin, should be classified as simple, chronic, stricture, and that they have their origin in an infection carried to the walls of the ureter from some distant focus such as diseased tonsils, sinuses, teeth, or gastro-intestinal tract.[14] This conception of stricture postulates that in the majority of cases ureteral infiltration is primary, and that the other urinary tract lesions so often associated with stricture, such as stone in the ureter, hydronephrosis, pyelitis, and pyonephrosis, are secondary.

As reported in my first paper on "Chronic Urethritis and Chronic Ureteritis Caused by Tonsillitis," read before the 1910 meeting of the Southern Surgical and Gynecological Association at Nashville, and as borne out by a fairly wide experience since that time, I have the proof that certain obstinate cases of chronic urethritis, which will not heal under the ordinary methods of treatment carried on over a long period of time, will promptly become absolutely well without further local treatment by getting rid of the distant focus of infection.

In the case of ureteral stricture it will probably take some years of experience before we can claim to have satisfactory

[8] Virchow's Arch. f. path. Anat., October, 1911.

[9] Zeitschr. f. Urol., 1912, III, 464.

[10] Diseases of the Kidneys, Ureters, and Bladder. Appleton, 1914, II, 354.

[11] Surgical Diseases of the Kidney and Ureter, 1901, II, 332.

[12] Proksch: Arch. f. Dermat. u. Syph., 1899, XLVIII, 224.

[13] Carey and Laird: A Peculiar Hypertrophy of the Prostate Accompanied by Ascending Infection and Cysts in the Ureter. Albany Medical Annals, July, 1904.

[14] Hunner: Chronic Urethritis and Chronic Ureteritis Caused by Tonsillitis. Jour. Amer. Med. Assn., LVI, 937, 1911; Treatment of Pyelitis. Surg., Gyn. & Obstet., XV, 444, 1912; Diagnosis and Treatment of Obscure Cases of Pyelitis and Hydronephrosis. International Clinics, 22, IV, 1912.

proof of the above thesis. Not only must we collect a large number of cases similar to Case 7, reported in the above paper, in which symptoms of ureteral stricture began soon after an attack of tonsillitis, but we must have a series of cases of ureteral stricture in which relief fails through a long period of treatments and is then obtained after the distant focus of infection has been discovered and corrected.

I have a few cases which have persistently showed no material improvement in symptoms until after removal of infected tonsils, or teeth, and another list of patients who have been dismissed as well after dilatation of the strictures or after getting rid of the pyelitis by dilatation of the stricture and lavage, and who have returned with their old symptoms after a fresh tonsillitis attack or after undergoing dental work.

Symptoms.—Pain is the most universal symptom of ureteral stricture and only in rare cases is it absent. To attempt to draw a pain chart of this affection one would need a diagram of the human frame extending from the diaphragm to the ankles. The most deeply shaded portion of this chart would center in the local area of ureteral inflammation, or in other words in the broad ligament region deep in the pelvis.

In my preliminary paper above mentioned as read in New York, I said: "It is comparatively rare to have our attention directed to localized symptoms due to the stricture. The patient usually complains of pain in the kidney region or of bladder symptoms or of both, and the stricture is found in the attempt to fathom the cause of the symptoms."

This statement was made after a study of my first 50 cases seen at a period when ureteral stricture as an entity was not in mind. Many of those patients came late when the kidney lesion was advanced and overshadowed other conditions. Since making that report and realizing the importance of ureteral stricture, better histories have been obtained, and it is rare to find a patient with stricture who does not complain of a pain or of a nagging discomfort in the site of the ureteral inflammation.

From this center of inflammatory discomfort in the pelvis the pain may radiate in any direction, upward toward the kidney, laterally into the hips or groin region, posteriorly, simulating a sacroiliac joint condition or a sciatica, and downward into the thigh and leg either posteriorly or anteriorly.

Next in frequency to the local ureteral pain is pain in the kidney region. This is probably at times a referred pain from the inflammatory area in the ureter, but it is usually due to overdistension of the pelvis of the kidney.

At times, when the renal catheter is passed and the wax tip and again the wax-bulb impinge upon the tender stricture area, the patient locates the pain in the kidney region. The kidney pain is usually elicited only by injecting the pelvis to overdistension. Frequently we get the history that the patient suffered at first with a more or less constant pain low in the abdomen, and that she later developed intermittent attacks of pain in the kidney region. Early in the patient's history these intermittent kidney attacks may come on only at the menstrual period or on getting the feet wet, or on having some unusual exposure, conditions that would favor the closure by swelling of an already narrowed area in the ureter. Later the pain in the upper flank or lumbar region may occur at frequent intervals or become as constant as the ureteral pain.

Occasionally the pain occurs first in the kidney region and develops later in the lower abdominal quadrant or region of the ureteral stricture. (See Figs. 2 and 3.)

Actual pain in the bladder occurs only in the exceptional case, and this is probably a referred pain from the kidney or ureter, as it accompanies the severe acute renal attacks simulating stone in the ureter in which there are likely to be both bladder and rectal tenesmus. Figure 4 illustrates such a case in which the two attacks of renal colic were accompanied by bloody urine and were controlled only by repeated hypodermics of morphin.

Discomfort in the bladder and frequency of voiding are not at all uncommon in the intermittent renal attacks, and these symptoms may be present in patients complaining only of the ureteral pain. One of my recent patients, not in the first one hundred, complained chiefly of a nagging desire to urinate.

Miss P., aged 51 years, had had multiple fibroids removed by Dr. Kelly 4 years previously. Three years later she had begun to have bladder symptoms and in September, 1916, she had returned to Dr. Kelly, who had failed to locate the cause. In May, 1917, she consulted me and in addition to her almost intolerable nagging desire to urinate, causing her to void from every hour to 3 hours in the day and to arise many times at night, I found that she complained of a dull pain in the left hip, of a pain high in the left lumbar region, and of a pain extending down the posterior left thigh into the calf of the leg. More recently she had developed an intermittent pain of less severity in the right hip and in the right side of the back. Investigation revealed a normal urine, a stricture of the urethra but a perfectly normal-appearing vesical, trigonal and urethral mucous membrane. The left ureter had such a dense diffuse stricture that I got by only on the third attempt in a series of 7 efforts within 10 weeks. I found a series of 4 dense areas of obstruction in the left ureter within 6 centimeters of the bladder, and that the kidney pelvis held only 6 c. c. The right ureter showed a dense diffuse stricture extending from the bladder to 9.5 cm. above, which could always be dilated; the kidney pelvis held double the normal quantity. Manipulation of the stricture area on either side caused her nagging desire to void, and manipulation of the left ureteral stricture caused pain in the kidney region, hip, and in the calf of the leg.

Urine examination.—The urine may be quite negative on repeated careful centrifuging and microscopic search. If there is an associated pyelitis, the urine shows the pathologic features and variations common to that condition. If the urine is not infected we may still find a few leukocytes or a few erythrocytes or both. These may come from an ulcer area at the site of the stricture, or we may find them increased in number after one of the acute kidney attacks, when they probably result from the trauma to the kidney pelvis. Figure 5 illustrates a unique case of symptomless hematuria apparently due to ureteral stricture.

Fever.—Chills and fever are common in the cases with urinary infection. A patient with an infection may go for weeks or months without chills or appreciable fever and, indeed, may be in apparently perfect health, or may suffer only from malaise and general depression. When there occurs any condition that

causes the stricture area to close the patient will develop fever (often with chills) pain and general prostration, such as are usually seen in acute pyelitis.

It is important to note that a patient may develop fever even of a high grade without infection of the urine and with only one ureter involved. The urine may be quite normal or there may be only a few erythrocytes. The kidney is enlarged, tender and painful. A few of the patients with sterile urine have shown a slight daily rise of temperature and these are likely to have nausea, headache, or other uremic symptoms.

One patient with a daily rise of temperature of 99° to 100° F. extending over a period of four weeks, and with a leukocytosis of 19,000, falling in the same period to 12,000, had a normal temperature and leukocyte count of 10,000 ten days after tonsillectomy.

Miss M., aged 22, nurse at the Hebrew Hospital, was awakened one Friday morning with a severe cramp-like pain in the right kidney region extending downward and forward to about the pelvic brim region. She had several attacks with severe pain, nausea and vomiting but kept on duty until the following Monday, when she reported to the superintendent of nurses that she could not continue with her work.

The first attack came on at the end of a menstrual period. For two nights before the attack she had been up frequently to void, and she had too frequent voiding up to the time of entering the hospital as a patient.

Drs. Adler and Hirsh were called to see the patient and because of the character and location of the pain and the presence of microscopic blood in the urine, they considered the case one of stone in the kidney or ureter. X-rays were negative and they called me into the case on October 20, the twelfth day of her illness.

Because of a daily rise of temperature to above 99° F. and a leukocytosis of 19,000 with a high percentage of polymorphs, we considered seriously the possibility of an appendicitis, with a high posterior position of the appendix to account for the pain phenomena. The presence of the above signs and symptoms without pus in the urine argued against a kidney lesion. Blood in the urine is not particularly rare in appendicitis. The patient's pain was more pronounced and more prolonged than one usually sees in appendicitis. She was rather stout, and considering the possibility that the X-ray might have missed a stone, I catheterized the kidney with a wax-tipped bougie. This came back without scratch marks. The meeting of the catheter tip with the kidney pelvis caused an increase of the patient's pain which she likened to that which she had had before. The injection of the kidney pelvis also exaggerated her former pain and the pelvis seemed to be rather smaller than normal, returning only 6 c. c. after being dilated to discomfort. This test of the kidney was followed by three days of intense pain instead of the usual few hours of discomfort caused by such an examination. The patient had to be chloroformed several times to control the pain. The temperature conditions and the leukocyte count remained about the same.

In view of the pain phenomena during catheterization and the great increase in pain after the catheterization, I became more convinced of the possibility of ureteral stricture, although there had been no apparent obstruction to the catheter. The possibility of stricture had been in mind from the first, when, on taking the history, I found that the patient had been troubled with her tonsils from time to time all her life, her last definite attack having occurred about 8 months previously. Another factor in favor of ureteral stricture was the presence of a dense stricture of the urethra. The tonsils appeared normal on examination.

One week after the first catheterization I again catheterized, this time using a wax-bulb. Again I could not detect obstruction, but the bulb must have had a beneficial effect for the patient did not have the severe pain reaction. Again the kidney pelvis seemed rather smaller than normal.

A week later it was time for the patient to menstruate and she was having increased pain in the kidney region. On catheterization with the wax-bulb catheter, I did not detect obstruction, but the wax-bulb, which was made of white wax and olive oil, equal parts, was partially mashed on withdrawal. A test of the kidney pelvis at this time when the patient was about to menstruate and was having increased pain in the kidney region, showed a capacity of 11 c. c. as compared with 6 c. c. on the two former tests. A week later, 5 weeks after the beginning of the patient's symptoms, my wax-bulb was definitely obstructed at a point about 3 cm. after entering the ureter, and on its withdrawal it was so hung in the stricture area that I feared it would be dragged off the catheter. Fortunately I had a bulb of pure wax on this occasion and it returned intact. After this catheterization the patient had a good deal of pain for an hour or two, due to the trauma incident to the dilatation of the stricture of the urethra and of the ureter. There was none of the increase of kidney pain as on former treatments. A few days later I again dilated the ureteral stricture with a wax-bulb of about 4 mm. diameter and then referred the patient to Dr. Lee Cohen for tonsillectomy. Dr. Cohen reported that on superficial examination the tonsils appeared healthy, but at operation they were found to contain large lacunæ of free pus.

Gastro-intestinal symptoms are common. They are probably of twofold origin, arising either as a central nervous system reflex or as a result of toxic absorption. We see the same phenomena in certain cases of stone in the ureter and in cases of blockage of the ureter from any cause.

The symptoms vary from a slight nausea or aversion to food to the most extreme nausea and vomiting sometimes so persistent that starvation is threatened. Gaseous distention is a frequent symptom. Occasionally there is complaint of rectal tenesmus and desire for stool. Pain just before or during stool is present in some cases, and is probably due to the passing of fecal matter over the tender peritoneum at the site of the ureteral inflammation. In Case 66 (see Fig. 6) the patient learned by experience that it was more agreeable to omit the meal preceding the dilatation and lavage; otherwise she lost it by vomiting. In Case 65 (see Fig. 7) the patient had suffered for a year with repeated attacks of diarrhœa and had lost 40 pounds in weight. Dr. Barker had found that she had an associated achylia gastrica and she had been given the dilute hydrochloric acid. After dilatation of her ureteral strictures her diarrhœal attacks ceased and she gained 11 pounds in 2 weeks.

Colitis may occur as a result of stricture. I recently referred a patient to Dr. Ernest Gaither because of much pain in the sigmoid region, through the left hip and in the upper flank, together with frequent stools in which she passed mucous casts. After a careful study of her case Dr. Gaither asked me if it could be possible that she was suffering from a ureteral stricture. That this diagnosis had not occurred to me in going over her symptoms was the more remarkable because she was a patient (Case 2) reported in my paper above referred to on chronic urethritis and chronic ureteritis caused by tonsillitis. A urethritis of 2 years' duration had resisted treatment for several months until her infected tonsils were removed. She had remained free from the urethral symptoms

for more than 6 years and had then begun to complain of the colitis which, after Dr. Gaither's suggestion, I found to be due to a dense stricture of the left ureter. Manipulation of the stricture area and injection of the slightly dilated kidney pelvis did away with all of her discomfort on the left side, and her colitis symptoms ceased after the second ureteral dilatation.

Mental Symptoms.—One of the remarkable results of this study has been to find that in a patient who may have one normally functioning kidney and only a slight degree of stasis on the opposite side there may supervene mental disturbances, headaches, nausea, fever, and a general picture usually associated with the uremic state. Figures 8 and 9 are from a patient who had a ureteral kink due to a fan-shaped band of adhesions which suspended her sigmoid to the posterior peritoneum just above the pelvic brim region. While in the recumbent position, taking a "rest cure," to many of which she had been subjected during the 20 years of her symptoms, she would take on weight and be comparatively well. As soon as she returned home and attempted to lead a normal busy life, the weight of the sigmoid displaced her ureter and again caused her "spells." These attacks consisted of a fever ranging from 99° to 100° F., malaise, headache, and a slight pain in the left lumbar region. The patient could "taste her fever" and tell by this peculiar reaction just where the thermometer would register within one-tenth of a degree. It is impossible to say what share toxemia played in her mental condition and how much of it was due to the disappointment and worry over her inability to get well. During her first 6 weeks under Dr. Barker's care it was considered essential to keep her in a barred room and have the constant supervision of a nurse.

The urine was quite normal and X-rays of the abdomen were negative. Dr. Barker asked me to investigate the left kidney because of the slight lumbar pain accompanying her attacks. After freeing the adhesions between the sigmoid and the left ureter peritoneum the patient made a prompt and full recovery and wrote me 6 months after the operation that she had gained 22 pounds.

Another case showing marked mental symptoms due to obstruction in one ureter was that of a physician who suffered for about a year with a morbid mental state entirely foreign to his usual buoyant disposition. Delusions and hallucinations worried him daily and, as in the case above reported, suicide became an urgent impulse. He was entirely relieved after Dr. George Walker had dilated a ureteral stricture.

Morbid Anatomy.—We have had opportunity to study the stricture macroscopically in about 15 operation cases. The inflammatory area varies from a slight annular thickening in the ureteral wall to a condition of diffuse cartilage-like thickening which may occupy several centimeters of the ureter and form a mass a centimeter in diameter. Multiple annular strictures are not uncommon.

The infiltration may be confined to the ureteral wall or there may be much periureteritis. Often at operation one cannot tell by palpation whether he is dealing simply with a stricture or with a stricture containing a stone.

I have had opportunity to study the stricture microscopically in only 3 cases (Cases 6 and 26) in which resection of the ureter and implantation into the bladder was performed, and in Case 50, in which we did a nephrectomy. The microscopic picture is one of chronic inflammation of all coats of the ureteral wall; the epithelium is changed from the transitional stratified type to a more squamous type and in one of the specimens there was an ulcer with loss of the epithelium.

Location of the Stricture.—The stricture is located in the broad ligament region or within 6 cm. of the bladder in by far the greater number of cases. The next most frequent location is at the bifurcation of the internal iliac vessels or about 8 to 10 cm. above the bladder. This is from 3 to 5 cm. below the pelvic brim. In both of these regions we have a group of lymphatic glands and at operation these are sometimes found enlarged.

Contrast these locations with the generally accepted view that ureteral stricture is congenital and occurs at the points of congenital narrowing, namely where the kidney pelvis enters the ureter, at the pelvic brim, and in the bladder wall area.

In the preliminary report on my first 50 cases, 12 were reported as bilateral. Since then in 3 of the 38 cases, reported as monolateral, the patients have returned with stricture on the opposite side. Of the 35 monolateral cases, 20 were on the right and 15 on the left. Of these 65 ureters with stricture the lesion has been located within 6 cm. of the bladder in 56, at the region of the bifurcation of the internal iliacs in 8 cases, and next to the kidney in 1 case.

In my second list of 50 cases, 15 were bilateral; and of the 35 monolateral cases, 22 were on the right and 13 on the left.

Of this second list of 65 ureters the stricture was located in the broad ligament region only, in 51, in the iliac gland region only, in 1, and in both broad ligament and iliac gland region, in 13. In many of the last group of ureters with strictures in both iliac gland and broad ligament regions the wax-bulb could be felt to jump through several stricture areas and it is probable that in these cases of multiple nodes of thickening there was a condition of diffuse stricture.

Effects of the Stricture on the Upper Urinary Tract.—Of the utmost interest is the relationship between ureteral stricture and other lesions of the urinary tract. This study has done much to explain the etiology of many urinary tract lesions concerning which we have had erroneous ideas in the past.

The profession has become accustomed to view slight dilatations of the pelvis and ureter as secondary to an infection of the urine. It is even held for the early cases with only slight dilatation and sterile urine that the dilatation was due to a previous infection.

This view, so generally held, is well expressed by Braasch:[1] "Any considerable degree of infection involving the renal pelvis and ureter will be followed by dilatation. This dilatation is not caused by mechanical obstruction, but is the result of changes in the tissues and consequent retraction in the walls of the pelvis and ureter. The dilatation may vary from a scarcely

[1] Pyelography: W. B. Saunders Company, 1915, 145.

recognizable irregularity of the calyces or ureter to complete destruction of the pelvis. Evidence of an inflammatory process which has once caused dilatation will rarely be entirely obliterated. Such inflammatory changes in the pelvic or ureteral outline may be the only evidence of previous infection. The character and degree of an inflammatory process can often be determined better by means of the pyeloureterogram than by any other method."

A priori one would expect the inflammation and infiltration to result in shrinkage rather than in dilatation of the tract.

While conceding that it is possible for the urine to become infected at the same time that the infection, which is to result in the future stricture, is laid in the ureter wall, I think these studies prove conclusively that the usual sequence is focal infection settling in the ureter wall, stricture formation, stasis and secondary infection of the urine.

Although we believe we have demonstrated that most cases of dilatation of the kidney pelvis are due to mechanical obstruction in the form of ureteral stricture, a surprising development has been the fact that many cases of ureteral stricture causing typical symptoms are not associated with a dilated pelvis, and when the pelvis is not dilated, it is often contracted. These patients with contracted pelves are usually the most sensitive to manipulation and the contracted pelvis is probably explained by assuming that the extremely sensitive stricture area keeps up a constant pain reflex, causing a tonic spasm of the musculature of the tract above. In some cases the urinary analyses, functional test, and pyelograms indicate that the shrunken pelvis is probably due to a secondary chronic interstitial nephritis. (See Fig. 12.)

Pyeloureterograms of these cases show that in some the ureter seems contracted as well as the kidney pelvis (Figs. 10 and 11) and that in others the pelvis is contracted while the ureter is slightly dilated (Figs. 6, 11½, 12 and 13). In some of these cases the pyelogram shows a larger pelvis than one gets by measurements. Figure 11 shows an apparently normal-sized pelvis, but the capacity tests showed a tolerance of about 4 c. c.

In several patients with normal-sized or contracted pelves when first seen, the general symptoms have improved under repeated dilatations, but the kidney pelvis has in the same time developed a slight hydronephrosis. The probable explanation of this phenomenon is that a tender ulcer in the stricture area has healed under treatment, thus releasing the reflex spasm, but that the stricture is dense enough to keep up partial obstruction in spite of the dilatations.

The 2 groups making up my first and second list of 50 cases, respectively, illustrate strikingly the effects on the upper urinary tract when ureteral stricture is neglected.

The first group of 50 cases consisted largely of patients seen incidentally at a time when ureteral stricture was not being looked for. In general, in this group of cases it is seen that there is a relationship between the lapse of time, increase in the size of the pelvis, and development of secondary infection. As already indicated above, the second group of cases shows many exceptions to this rule.

In the first group of 50 cases there was no report on the condition of the urine in 6 cases. I have notes on cultures from 27 of the remaining 44 cases. In 9 of these the urine was sterile. Of the 17 cases in which there was no mention of cultures, the urine from the kidney was free from leukocytes in 7, and was presumably sterile, which would make 16 out of 44, or over 33.3 per cent of the reported cases, sterile. In 10 of these 17 cases without mention of cultures there was pus in the specimen catheterized from the bladder, and in 7 of these the kidney specimen is also reported as yielding pus; so that possibly all 10 cases were infected.

In the 16 non-infected cases the average age was 38 years, the average duration of symptoms 2½ years, and the average size of the kidney pelvis in 10 of the cases was 19 c. c., 3 of these holding 8, 11, and 12 c. c. respectively, and the others holding from 15 to 30 c. c. In one exceptional case with symptoms of 4 years' duration the pelvis had reached a capacity of 360 c. c. without becoming infected. In 5 cases there was no note on the pelvis capacity.

In the 18 infected cases the average age of the patients was 35 years, the average duration of symptoms was 4 years, and the average capacity of the kidney pelvis in 15 cases in which a record was made, was 98 c. c. In 4 of these 15 cases the pelvis was of normal capacity, 7 to 8 c. c., showing for the 11 dilated cases an average capacity of 130 c.c. In the cases in which both pelves were dilated the capacity is figured on the larger pelvis only.

In the 18 infected cases of the first group the colon bacillus was grown in 13, in 5 of these from both kidneys. In 4 cases a staphylococcus only was grown, in 1 case a pure typhoid culture was obtained (nephrectomy for large pyonephrosis soon after typhoid fever). In one of the colon bacillus cases B. coli was grown from one kidney and a staphylococcus from the other, making 5 cases in which a staphylococcus was grown.

In the second group of 50 cases all were studied bacteriologically. In 11 there was an infection, in 8 with the colon bacillus (4 bilateral), in 1 with a staphylococcus, in 1 with a streptococcus, and in 1 an unidentified organism. Taking into consideration the 4 bilateral infections there were 15 kidneys involved, and only 4 of these showed dilatation, measuring respectively 15, 14, 20, and 15 c. c. Three of the kidneys measured 7.5 c. c. each and the remaining 8 measured 6 c. c. or less.

In this second group of 50 cases, 15 of which showed bilateral stricture, notes were made on the pelvis content of 63 kidneys. Of these, 22 were normal and ranged in size from 6 to 8 c. c., in 8 the capacity was considered less than normal, the pelvis measuring 5 c. c. or less. In 24 kidneys dilatation was demonstrable and if we exclude the two dilated pelves in Case 65 (see Fig. 7), the remaining 22 pelves showed an average dilatation of 16 c. c. in marked contrast to the many large pelves in the first group.

The average age of the patients in the second group at the time of consultation was 34.5 years and the average duration of symptoms was 5.3 years; but if we separate from this group 7 cases of unusual duration averaging 19 years, the average duration of the remaining cases was 2.6 years.

Influence of Ureteral Stricture on Stone Formation.—One of the most interesting side-lights on ureteral disease furnished by this study has been the revelation of the probable cause of most ureteral stones. In operating for a ureteral stone and finding it encased in dense infiltration tissue, we have heretofore considered the inflammatory area as due to the irritation of the stone. At present we have abundant evidence to indicate that the stone results from urinary salts being deposited on the inflamed surface of the stricture area.

Every surgeon sees an occasional case in which a minute stone or a nest of minute stones is found encased in a dense stricture area. It is quite evident that such small stones were not formed in the kidney and then stopped in a normal ureter, nor were they formed in a normal ureter. Such small stones could easily be passed entire through a normal ureter.

There can be no doubt that an occasional stone formed in the kidney is caught in a normal ureter. On the other hand, I believe that some stones found in the kidney were originally formed on the site of a ureteral stricture and after sufficient dilatation of the tract above, these stones had floated up into the kidney where they had increased in size. Figures 14 and 15 illustrate 2 such possible cases. Figure 16 illustrates another possible sequence, *viz.*, that a stone forming in a stricture area may develop a pressure above sufficient to force it out of the stricture and into the bladder. In this case the stone was spontaneously passed from the bladder. In Case 23, the patient had suffered with bladder discomfort and incontinence for 9 months. A stone, 15 mm. in diameter, was removed from the bladder. The X-ray revealed another small shadow in the left broad ligament region, and investigation with the wax tip and wax-bulb catheter demonstrated this to be a venous phlebolith, but the patient had 2 strictures in the broad ligament region and a left colon bacillus pyelitis with a kidney content of 21 c. c.

In Case 71 the patient, aged 37 years, had suffered for 8 years with left renal colic, the attacks becoming so severe recently that they required several hypodermics of morphin for their control. The urine was smoky with blood and contained pus and a colon bacillus infection. On passing a catheter prepared with small wax rings at intervals of 5 cm. for locating the possible ureteral stone, and with one large wax-bulb to dilate the ureter, the catheter returned with scratch marks on all the rings within 20 cm. of the tip. The eye of the catheter contained a particle of stone and the large wax-bulb had embedded in its surface about a dozen particles of stone. The patient was completely relieved of her severe colic attacks and subsequently had several dilatations of the stricture area until her symptoms were relieved. She had the contracted type of pelvis, holding but 4 c. c. Scratch marks were never obtained after the first treatment, which seemed to have entirely cleared out the incipient stone formation.

One of the strongest arguments for the relationship between stone formation and stricture is the finding of bilateral symmetrical stricture and a stone in one of these stricture areas, as has occurred in 4 of my cases, 2 of which are illustrated in Figures 17 and 18. A large stone (see Fig. 19d) was removed from the right broad ligament area in Case 9, 5 years after I had been dilating strictures in the right broad ligament area and iliac gland region and in the left broad ligament region. The obstruction by the right broad ligament stricture and its stone had made a large dilatation of the right ureter in which at operation the stricture at the iliac gland region was seen to form a dense annular narrowing.

O. S. Fowler, of Denver,[16] has called attention to the possible importance of stasis in the etiology of stone formation. He attributes the stasis to a kink in the ureter due to prolapse of the kidney as demonstrated by the method he has done so much to develop of taking pyeloureterograms in the erect posture. Some of his roentgenograms, however, reveal ureters which are dilated below the point of kinking as well as above and I would interpret them as being cases of unrecognized ureteral stricture low in the channel causing a dilatation of the ureter and kidney pelvis, and a prolapse of the heavy hydronephrotic kidney, causing apparent kinks in the upper ureter. I have several cases of stricture of the ureter showing this kidney prolapse and apparent kinking of the ureter, but they all show the ureteral dilatation down to the stricture area as illustrated strikingly in Figures 2 and 3.

Influence of Ureteral Stricture on the Pyelitis of Pregnancy and of the Puerperium.—Of my last seven cases of pyelitis developing during pregnancy or soon after delivery, 3 being bilateral, I have been able to demonstrate stricture of the ureter in all but one case. This is probably a much higher percentage associated with stricture than we would find in a larger series. In most of such cases the stricture is probably present before the pregnancy and the added congestion of the tissues after conception sets up a slight hydronephrosis which becomes infected during the pregnancy or immediately after.

Some of the cases of pyelitis of pregnancy due to stricture clear up spontaneously after labor after the congestion and edema incident to pregnancy have disappeared. Others continue as a chronic pyelitis until treated. Undoubtedly most of those cases of pyelitis of pregnancy which clear up spontaneously or after a few irrigations with silver solution do so because a stricture is not present, or because the slight dilatation of the stricture incident to passing an ordinary renal catheter is beneficial.

That a case may clear up spontaneously in spite of ureteral stricture is illustrated by a patient referred by Dr. Barker (Figs. 12 and 13) for attacks of renal colic associated with chills and high temperature. Fourteen years previously Dr. Kelly had been compelled to perform an abortion for this patient because of a bilateral colon bacillus pyelitis. She had no special treatment for the pyelitis, returned home with an infected urine, and had recurring attacks of pyelitis symptoms on the left side during the intervening 14 years and on the right side for 9 years. Examination revealed very dense bilateral strictures with the kidney pelves smaller than normal; the ureters showed very slight dilatation. The urine contained

[16] Ureteral Obstruction Causing Urinary Stasis; A New Etiology in Kidney Stone, etc. Jour. Amer. Med. Assn., 1914, LXII, 367.

many hyalin and granular casts and a few pus cells which were proved to be coming from the left kidney. There was no infection. It could very well be argued that this patient had no stricture at the time of her pregnancy and that the pyelitis infection resulted in later stricture formation. My experience with such cases leads me to take the contrary view, particularly as she had suffered all her life with tonsillitis, and because she obtained no permanent improvement from ureter stricture dilatations until after the badly diseased tonsils were removed.

Diagnosis.—The diagnosis of ureteral stricture depends upon the history, urinary examination, palpation of the abdomen with special reference to the kidney and ureter regions, palpation of the ureters through the vagina or rectum, cystoscopy, catheterization of the ureters by specially prepared catheters, and roentgenography.

The features obtainable from the history have been discussed above under the section on symptoms. Experience has taught that we should suspect stricture in any patient complaining of obscure abdominal symptoms, particularly in the lower abdomen and accompanied by referred pain in the hips and thighs. In addition we usually find that the patient has a history or shows evidence of tonsillitis, sinusitis, or bad teeth.

After a relatively wide experience in the diagnosis of such obscure cases I have learned to lay a good deal of stress upon a history of previous abdominal or pelvic operations from which the patient has failed to obtain relief. In the second group of 50 cases 34 operations had been done, 7 of which seemed to have been necessary and 27 unnecessary and not followed by relief. Four of the patients had each had 3 operations without relief from the original symptoms. A more recent patient, 36 years of age, with bilateral stricture, had spent a good portion of the previous 18 years in hospitals, having been submitted to 8 operations, 7 of them abdominal. Figures 10, 11, 11½, 15, 20, 21, 22, 23, and 35 illustrate cases of patients who had been operated upon previously without relief from the chief symptoms.

Of course one cannot judge accurately as to what past operations may have been necessary, and we must keep in mind the frequency of ureteral stricture and the possibility that it may have been present with appendicitis, gall-stones, floating kidney, intestinal adhesions, ovarian disease, uterine misplacements, or any of the numerous lesions for which the former operations were done. Three of the above 27 operations listed as unnecessary were performed by myself, 2 for appendicitis, in which a note was made at the operation that the appendix appeared normal, and one for relaxed vaginal outlet and cystocele. In this last case (No. 58) pus and blood were present in the urine and were attributed to a severe trigonitis which in turn was supposed to be due to the cystocele. The patient complained chiefly of a nagging pain in the left broad ligament region, frequency of micturition, and partial incontinence. With the cystocele operation we puckered her sphincter muscle region and cured her incontinence, but she experienced no other relief from the operation and a year later we found that her symptoms of left-sided pain and pollakiuria were associated with bilateral stricture and colon bacillus pyelitis. We have dilated the strictures and cleared up her pyelitis by lavage and she still has a nagging trigonitis and is planning to have her bad tonsils removed.

It is of great importance to remember that symptoms due to ureteral stricture often begin in early childhood (see Figs. 24, 25, 26, 30, and 36).

Urinary Examination and Diagnosis.—In the section on symptoms we stated that the urine may be quite normal. This has undoubtedly been a source of frequent error in the past. Given a patient with symptoms that suggest trouble in the urinary tract, we have been willing to exclude the urinary tract on finding a normal urine. If the patient's symptoms strongly suggested stone in the ureter we have had X-rays taken. We have the word of the X-ray experts that they miss anywhere from 15 to 30 per cent of ureteral stones—a fact that alone should call for an examination with the wax-tipped catheter in all cases of suspected stone. With the facts that the unassisted X-ray will miss all ureteral stricture cases and that the urine may be normal, we have thrust upon us a duty heretofore neglected, namely, to have all questionable cases investigated with specially prepared catheters and perhaps with the thorium X-ray.

In the second group of 50 cases there were 39 without infection of the urine. We have definite notes on the examination of the urine catheterized from the bladder in 36 of these non-infected cases, and it was normal in 26 cases, contained red blood cells in 5 cases, leukocytes in 2 cases, and both red and white cells in 3 cases.

Palpation.—There is often tenderness in the renal region and the kidney may be somewhat enlarged and very tender during one of the acute attacks of pain. The usual phenomena of an intermittent hydronephrosis may be demonstrated if this condition be present.

Palpation of the ureter over the pelvic brim may elicit tenderness and a desire to void. This tenderness over the region of the pelvic brim has led to many useless appendix operations. With both conditions in mind when palpating, one can usually differentiate between them, as the patient will indicate that her usual area of pain is deep in the pelvis, back of the symphysis, although one may elicit more tenderness over the pelvic brim on abdominal palpation and mistake it for an indication of appendicitis.

Vaginal palpation will show the maximal tenderness to be in the broad ligament region in a vast majority of cases, and one may actually palpate the node of thickening which in some cases cannot be differentiated from stone in the ureter.

One may easily mistake the ureteral tenderness for a painful ovary. In Case 56 the patient, then aged 40 years, consulted me first 5 years ago complaining of some of the disturbances of the menopause and a nagging pain low in the left pelvis. She had been advised to have a left oöphorectomy. I found that the left ovary, while apparently extremely tender, was of less than normal size, feeling like a senile fibrous ovary. I advised against operation. She recently consulted me again, having suffered intermittently with the same pain

and finding it recently much exaggerated after a long automobile ride. She had also developed marked pain in the left kidney region and feared that she might be developing cancer.

I suspected ureteral stricture and found on bimanual examination, just as I had noted 5 years previously, that the left ovary was small and hard and apparently very sensitive. By carrying the ovary medianward and isolating it from the broad ligament region I found that it was not at all tender. Then by dropping the ovary and palpating the ureter by itself, I found the latter to be the seat of pain and tenderness. Two dilatations of her left ureteral stricture have resulted in a most satisfactory clearing up of symptoms of 6 years' duration. There was a draining abscess at the root of one tooth and she was referred to her dentist to have X-rays and the necessary dental work attended to.

Cystoscopy is usually quite negative, but in the occasional case in which the stricture is near the bladder wall there may be redness and edema about the ureteral orifice suggesting the picture seen with a low ureteral stone. One of the most suggestive points in cystoscopy is the finding of a urethral stricture when preparing the urethra for the cystoscope. Although stricture of the female urethra is common after a gonorrheal infection I have learned by experience to give its presence considerable weight in the diagnosis of a suspected ureteral stricture. We have neglected to make any note on the condition of the urethra in many cases, but in the last 50 cases a note was made in 28, and in 27 of these there was a stricture of the urethra. I think complete notes would have shown at least 60 per cent with a urethral stricture. It is curious that definite annular stricture of the urethra may be present in some cases without bladder symptoms and with a perfectly healthy looking urethral mucosa.

Catheter Test.—The crucial test in diagnosing ureteral stricture is made with the wax-bulbed catheter. This, with other instruments used in getting past and treating stricture, will be described under the section on treatment. I do not consider obstruction to an entering catheter as diagnostic of ureteral stricture. Repeated obstruction at the same point is certainly suggestive, but for a positive diagnosis I depend entirely upon the obstruction or "hang" of the wax-bulb on withdrawal. Obstruction of the tip at a certain point in the ureter and then obstruction of the entering bulb at the same point, and the going by of the bulb with a jump and a sense of scar-tissue grating, are points upon which the experienced cystoscopist may rely with a fair degree of safety; but there are many conditions which may obstruct a catheter on entering, and it is better to depend upon the "hang" of the bulb, its jump, and scar-tissue grating sensation as it comes through the stricture area on withdrawal. In the case illustrated by Figures 8 and 9, I did not get by the obstruction until the third attempt at intervals of 10 days, and then only by changing the position of the patient from the knee-chest to the right lateral position while using a considerable amount of force on the catheter with the stylet in. I considered this a stricture case because of the great difficulty in getting by, but after getting the thorium X-ray and before development of the plates, I said the case was not one of stricture because on withdrawal of the bulb there was only the faintest obstruction at the pelvic brim region, just as one can detect as the bulb comes through the bladder wall and again as it comes through the sphincter urethræ. It has been a source of great satisfaction to my associates in this work and to myself to find at operation in a number of cases how accurately the wax-bulb has located the position and the number of strictures; and how, in other cases like the one just cited with ureteral obstruction but no "hang" of the bulb on withdrawal, the operation has explained the cause of the obstruction.

Dr. Holmes, resident gynecologist at The Johns Hopkins Hospital, asked me to test a ureter in which he had found great obstruction to the wax-tip and wax-bulb catheter on entering, but no appreciable obstruction on withdrawal of the bulb.

Mrs. H., aged 43 years, had been operated upon some several years previously, the right ovary being removed. She complained of a constant pain in the lower right quadrant and at times when this got worse she also had pain in the right back and across the abdomen. I found, as Dr. Holmes had done, that it was very difficult to pass a renal catheter through the pelvic portion of the ureter, but that the wax-bulb returned without appreciable obstruction. The passage of the catheter caused a pain like her former pelvic pain, and injection of the kidney pelvis induced her old pain in the right back. At operation we found that the patient had a much infiltrated and adherent appendix. The former ovarian operation had been followed by a dense adhesion of the sigmoid to the region of the right ovarian vessels. The pelvic peritoneum was reddened and thickened and on opening this and exposing the right ureter, I found that it had left the peritoneum and was adherent to the outer pelvic wall as a result of a marked periureteritis. With a tape under it the ureter was lifted and explored to its entrance into the bladder without the discovery of any local infiltration. It is probable that the hooked-up sigmoid or the appendix, or both, caused a chronic pelvic peritonitis, and when the pelvic inflammation grew worse, there was enough swelling of the ureteral tissues to cause partial stasis and her pain in the kidney region.

In a recent case in which there was much discussion as to the diagnosis, operation revealed a ureteral stricture where our wax-bulb had located it, at a point beneath the uterine vessels, or 8 cm. from the external urethral orifice.

Mrs. W. was referred to me in March, 1917, by Dr. Harry Adler, with a probable diagnosis of gall-stones and appendicitis. Roentgenography had shown small shadows in the gall-bladder region and a bismuth stasis in the ileocecal region after 24 hours. Because of their multiplicity and lack of special characteristics in her symptoms I decided to test for ureteral stricture. This was definitely located in the broad ligament and some of her former symptoms were duplicated by injection of the kidney and manipulation of the stricture. On the day of operation and after the patient was anesthetized, I passed a No. 8 renal catheter prepared with a large wax-bulb, and left this in place during the operation.

Because of a wide diastasis of the rectus muscles a long midline incision was made. A definitely diseased appendix was removed, and ileocecal bands and sigmoid adhesions were freed. Gall-stones were palpated and before making the separate gall-bladder incision I investigated the ureter. With its contained catheter it was easy to palpate the ring of thickening in the broad ligament region. The posterior peritoneum was opened to expose the ureter and an assistant then removed the catheter. It slid out

easily until the bulb caught in the stricture area, when the tug displaced and straightened the lower half of the pelvic ureter. After the catheter was out my assistant easily palpated the stricture area.

It is important to remember that one may pass the ordinary renal catheter of large size without detecting a stricture. The presence of a hydronephrosis should make one suspect stricture and test for it with the wax-bulb. In the case of a pyelitis in which catheterization is followed by fever or a chill, one should suspect stricture and should wait at least a week before making the next test with the wax-bulb. Some cases without infection show a marked reaction after catheterization, having an elevation of temperature and an unusual amount of pain and sometimes nausea and vomiting. Any of these unusual reactions after the passage of the ordinary catheter should prompt one to test for stricture. In some of my pyelitis cases I have not suspected stricture until the patient failed to show improvement after repeated lavage. Figure 6 shows a case in which I had washed with silver nitrate solution 6 times and then only had suspected stricture because of the lack of improvement in the pyelitis.

In Case 49 (Fig. 24) I diagnosed stricture from the correspondence about the case, this indicating that the patient had but one kidney and that this was the seat of an infected hydronephrosis. She was getting lavage treatment but frequently had high fever, chills and prostration after the treatment. My correspondent also informed me that she had lost one kidney by operation at the age of 22, after suffering with intermittent hydronephrosis attacks since babyhood, and that she had always been subject to tonsillitis.

Roentgenography.—The taking of pyeloureterograms is not necessary in making a diagnosis except in the few cases in which we cannot go by an obstruction in the ureter. Under such conditions if we can pass an X-ray catheter with a whistle tip to the point of obstruction, we can often get the contrast solution to go beyond the obstruction and intensify a stone, if it be present, or outline the character of the obstruction and the condition of the tract above.

In Case 50 (Fig. 15) we failed on several occasions to get enough thorium past the stricture to get a shadow in the kidney pelvis. Urine with pus and blood was coming from the kidney.

In a recent case (Fig. 23) we were arrested at an obstruction on two occasions and then had the nature of the case cleared up by injecting thorium beyond the stricture into the ectopic kidney. Later we successfully dilated the stricture and gave the patient relief from symptoms of 15 years' duration.

Although the X-ray is not necessary in making a diagnosis in most cases of ureteral stricture and although I refused to use it in my early cases when we were dependent on collargol with its dangers, I now use it in most cases for the satisfaction of its confirmatory value and because I find that thorium may be used with impunity if employed with judgment.

Figure 27 is from Case 16. The patient died in uremia without passing urine for 12 days after this thorium picture was taken. I attribute the death not to the use of thorium but to the treatment of both sides at the same time, that caused swelling and closure of both ureters. During the lavage of her kidneys one at a time at intervals over the previous 4 years she occasionally had a severe reaction—fever, chills, nausea, vomiting, headache, and prostration—and this should have been a warning not to traumatize both sides at one time.

For work on the male subject with the unavoidable restriction in the use of instruments, the pyelogram will prove one of the best aids in diagnosis of ureteral stricture.

The ureterogram is of great value in deciding whether a small shadow on the unaided X-ray plate is within the ureter or whether we are dealing with a phlebolith. This point can usually be easily settled in women by using the wax-tipped and wax-ringed catheter. In using the radiogram method the opaque catheter or the styletted catheter should be photographed in the ureter. Many stones in the ureter are in the broad ligament region, and this is true for many phleboliths. Hence, it is safer to use the bifocal method, as a phlebolith in the uterine vein may be superimposed on the renal catheter and with a flat plate we cannot make a certain interpretation.

Of even greater value is the use of these methods to differentiate ureteral stricture from supposed stone in the ureter when the unaided X-ray has shown a shadow in the ureteral region. Many surgeons have had the uncomfortable experience of operating on at least one of these cases. The patient complains of symptoms suggesting stone in the ureter, the urinary examination confirms this view, and the X-ray shows a shadow in the ureteral region.

Figure 28 illustrates a case of pyelitis occurring 4 months after the birth of a first child, and in which full investigation changed the diagnosis from a right pyelitis due to stones in the ureter to bilateral stricture with right pyelitis. Figure 29 illustrates a case referred by Dr. Hugh Trout of Roanoke, in which I was fortunate enough to get by her dense stricture with the X-ray catheter and demonstrate the extra-ureteral position of the stones. Figure 30 represents an X-ray plate brought with a patient referred by Dr. E. L. Mortimer, as a case of left pyelitis due to stone in the ureter. Investigation failed to show scratch marks and demonstrated that her bilateral pyelitis following childbirth was due to stricture. Figure 31 illustrates the case of a patient with a stone in the appendix and gall-stones, the symptoms being due to ureteral stricture.

Diagnosis from Tuberculosis.—Great care must be exercised not to mistake tuberculous disease of the ureter for ordinary stricture. In 1901, I did a resection of the ureter and implantation into the bladder on a patient who had much pus in the urine, and an impassable thickening in the lower end of the right ureter. One examination had shown no tubercle bacilli. The true diagnosis was revealed a few months later, when I was called to operate on the patient for intestinal obstruction, which was found to be due to tuberculosis. The tuberculous kidney was removed later and the patient made a good recovery.

In October, 1915, Dr. Charles Austrian referred to me a young unmarried woman, 22 years of age, who had been having a great deal of pain in the left pelvis and left kidney region, and bladder symptoms for about two years. She had had ton-

sillitis since childhood. She had had two operations, a dilatation and curettage, and a suspension of the uterus and appendectomy. After one successful dilatation of an obstruction low in the left ureter and the demonstration of a pyelitis, I was unable on several occasions to dilate again. I was planning to expose the ureter extraperitoneally and do a retrograde dilatation, when at a final cystoscopy I was impressed with the large amount of edema about the ureteral orifice and made a second search for tubercle bacilli, which resulted in demonstrating them in large numbers. Nephrectomy for tuberculosis resulted in a restoration to health.

Figure 32 illustrates a case in which I had successfully relieved the uncomfortable symptoms by dilating the stricture and in which I first suspected tuberculosis because the patient's large amount of pus failed to diminish after repeated lavage. My suspicions were increased when on getting this pyeloureterogram we saw the irregularly eroded character of the pelvic outline. A search for tubercle bacilli was positive.

Differential Diagnosis.—It may be well to close this section on diagnosis by giving a list of various diagnoses made upon these patients although their symptoms were chiefly due to ureteral stricture. *Urinary tract:* cystitis, pyelitis, pyelitis of pregnancy and the puerperium, pyonephrosis, floating kidney, hydronephrosis, stone in the ureter, chronic Bright's disease. *Genital tract:* pelvic inflammatory disease, ovarian disease. *Gastro-intestinal tract:* various functional disorders of the stomach and intestines, chronic peritonitis, intestinal adhesions, sigmoid adhesions, colitis, chronic pancreatitis, gallstones, appendicitis. *Joint and nerve conditions:* lumbo-sacral and ilio-sacral joint pains, neuralgia of the sacral plexus, sciatica. Mental disorders.

Treatment.—The chief end sought in treatment is the relief of symptoms, and in the infection cases a urine freed from infection, and in all cases suitable for dilatation such a thorough opening of the stricture area that there will be no recurrence.

There are very few cases in which we cannot at least ameliorate the symptoms, and fortunately we can relieve the patients, to a large extent, if not entirely, in the majority of cases. There are very few cases of pyelitis in which we fail to do away with the infection. I believe that time will demonstrate that in many cases we shall not get a permanent dilatation of the stricture, and complete relief of symptoms until we have eradicated the original focus of infection.

The ideal treatment for stricture of the ureter is by dilatation from the vesical approach. Naturally those whose work is confined to women and those who use the Kelly speculum have a great advantage in treating this disease. Various forms of operative cystoscopes and ureteral instruments have been devised by Bransford Lewis and others, which make it quite possible to do much effective work from the vesical end in the male.

My work being confined to women, I shall speak only of the methods which I have used in treating stricture, these having been largely developed or suggested by Dr. Howard A. Kelly.

A glance at Figure 33 will show the simple instruments which I use, entirely with the tubular speculum of Kelly. As a rule I use the olive-tip catheter (Nos. 7, 8, and 9) carrying a wax-bulb 8 to 10 cm. from the wax-tipped end (b). Not infrequently, when the olive-tip catheter refuses to pass, a round-tip (a) or a whistle-tip (c) will engage in the lumen and go by the stricture. One may use the ordinary whistle-tip catheter with the wax-bulb or the whistle-tip catheter with a gradually increasing diameter, which Garceau devised for the special purpose of making functional tests (c). This Garceau catheter (Nos. 11 or 13) gives a fairly good dilatation without the addition of the wax-bulb.

At times these catheters engage in the lumen of the stricture area better with the wire stylet left in for stiffening, and at times a slight withdrawal of the stylet will result in success when the catheter has seemed permanently obstructed.

In case of failure to get by with any of these forms of flexible catheter, it is sometimes possible to make the first entrance with the metal searcher (f). By slightly curving the last centimeter of the metal searcher, one can gently rotate the angled handle, thus giving the tip a variety of axes, in one of which it will engage and pass the stricture where the more flexible instruments will meet a pocket or shelf of mucosa and be permanently obstructed. Usually after getting through the stricture area with the olive-pointed metal searcher, one can withdraw the searcher and immediately get by with one of the flexible catheters which has previously been held; or one can follow the metal searcher with the metal bulb dilator (g), which has a slightly curved olive-tip, followed by a metal bulb, 3 mm. in diameter, which gives such thorough dilatation that the flexible catheter is quite certain to pass. At later treatments, where a still greater dilatation is desired, the metal bulb dilator 5 mm. in diameter (h) may be used. It is seldom necessary to use this large metal bulb, for one can easily use the flexible catheters with a wax-bulb as large as 6 mm. in diameter. In using these very large bulbs it is well to see that the bulb is of a perfectly spindle shape, having no abrupt shoulder to catch on the stricture.

In our earlier work in testing for stone in the ureter we always used beeswax (the cera flava or cera alba of the pharmacopœia) mixed with one-third or one-half of sweet oil, but I found that this softens the wax to such a degree that it is partially crushed on coming to a narrow orifice, or later on meeting the stricture; somtimes on withdrawal of the catheter, a part or all of the wax is left on the proximal side of the stricture. For these reasons I have given up the mixed wax and oil and use only the pure beeswax. This is soft enough to get a good impression from a stone, and it is firm enough to hang to the catheter and not be crushed under any ordinary condition of obstruction.

At times on failing to get any of the above instruments to engage in a stricture, I have succeeded in making the first dilatation by using the whalebone filiform searchers (e). Usually the first two or three searchers catch in the mucosa just as the other instruments have done, but after introducing three, four, or five filiforms, one can by careful manipulation get one

of them through the stricture lumen, and then on further manipulation the others can be made to follow.

After dilatation with from two to four filiforms, one can withdraw these and pass the renal catheter with the bulb, but it is generally safer to leave one or two of the filiforms as a guide and pass the metal searcher or the flexible catheter alongside the filiforms to engage the stricture lumen before the guides are withdrawn.

Before beginning work with the whalebone filiforms one should have sterile hands or put on a sterile glove, for these have to be grasped close to the speculum at a portion of the filiform that later enters the bladder or the ureter.

Finding by experience that the whistle-tip catheter often engaged and passed a stricture after the pointed tip had failed, and finding that the metal searcher with a slightly curved tip was a most useful instrument, apparently because a rotary motion enabled the curved tip to pick up different axes of direction in the lumen, I combined these two ideas of a beveled tip, and a fulcrum to throw the tip into various axes, by using the wax on the end of an ordinary pointed-end catheter, in a fashion to make a corkscrew or spiral-tip catheter (see Fig. 34). I have been successful in passing strictures with this spiral tip after failing with all other instruments. One can feel the rotation of the catheter as the spiral tip finds its way through the stricture.

Precautions in Technique.—Experience has taught a few precautions which should be observed in treating stricture. If the history points to the probability of bilateral stricture, never investigate both sides at the first examination, and in later treatments do not dilate both sides at one sitting until you are certain that the strictures are well dilated and that the kidneys will have drainage after the trauma of treatment. Case 16 (Fig. 27) emphasizes these points.

Do not investigate or treat a stricture too frequently. It seems to take about 10 days in the average case for the traumatic edema of the early investigation to subside. This is particularly true if the last effort to dilate was a failure, for in such a case there is usually more trauma than in the case in which the catheter and dilator pass on the first effort. If the patient is away from home and under the expense of hospital treatment she may be restive at the surgeon's apparent inactivity, and it is well to explain that greater haste is likely to defeat the desired end. For the patients who return to my office or to the dispensary for treatment I prefer to fix the interval at from 2 to 4 weeks or longer, according to the response.

In the pyelitis cases in which we are anxious to combine the pelvic lavage, we may observe the 10-day interval for the first two or three treatments or until we are quite certain of a good dilatation, then we may use lavage twice a week, employing a small catheter (No. 5 or 6) and once in 10 days or 2 weeks using a No. 8 or 9 catheter with the large wax-bulb for dilatation.

In any case do not use a large bulb for the first dilatation. A 3 or 3.5 mm. bulb will usually pass without splitting the mouth of the ureter and without too much trauma to the stricture area, although this small bulb may split the stricture and cause some bleeding. It is not uncommon in the pyelitis cases to have a partial closure of the stricture area after the first one or two dilatations with the small bulb; this being especially true if these treatments are at short intervals.

The use of large bulbs measuring 5 to 6 mm. is not without its danger even after the patient has had repeated treatments. In Case 35, the patient who had only one kidney (see Fig. 35) was ready to be dismissed as well when we undertook to make a final large dilatation. This evidently split her stricture area for she had intermittent suppression and passage of ureteral clots, and was in a precarious condition for several days until the bleeding stopped.

In two of my cases the use of too large a bulb or perhaps the presence of a too abrupt return shoulder on the wax-bulb resulted in great difficulty in extricating the catheter, and on its return the shoulder carried a complete collar of mucous membrane torn from the stricture area. This of course represents an undesirable degree of trauma in the stricture area.

One must use care in the amount of force exerted in pushing the catheter in. If the stylet is still entirely in the catheter the end of the catheter may go through the ureter. This evidently happened in Case 36, and a silver nitrate injection of 1:1000 strength was left in the extraperitoneal tissues of the pelvic brim region. This caused much pain of a peritonitic character which persisted for nearly a week but with no apparent permanent damage. In Case 29 the catheter went through the bladder portion of the ureter, penetrated the bladder mucosa, and curved back into the bladder.

If the stylet has been partially withdrawn there is probably no danger of the end of the catheter penetrating the ureter, but forcible pushing may result in penetration of the catheter wall with the end of the stylet and probable puncture of the ureteral wall, as occurred in a recent case without observable results.

Results of Treatment by Vesical Approach.—What can we hope for in the simpler non-operative forms of treatment? In the cases without infection and without much renal disturbance we can look for cure, if distant foci of infection have been removed. This was the result in 8 of my first 50 cases. The case numbers, the duration of symptoms, and the pelvic contents before and after treatment are shown in the following table:

Case No.	Duration of symptoms	Pelvis before treatment	Pelvis after treatment
15	A few weeks.	30 c. c.	3 years later, 15 c. c.
17	5 years.	22 c. c.	2 years later, 9 c. c.
21	2 months.	15 c. c.	5 months later, 7.5 c. c.
22	5 years before passing ureteral stone.	28 c. c.	5 months later, 10 c. c.
27	2 years.	30 c. c.	Not seen after 3 treatments and complete relief of symptoms.
28	9 years.	15 c. c.	3 years later: No further attacks. Pregnant for 3d time since treatment.
35	18 months.	22 c. c.	4 months later, 10 c. c.
40	3 months.	40 c. c.	Not seen after two treatments.

Since presenting the above table in my preliminary report of the first 50 cases, one patient (Case 15) has returned with renewed symptoms on the left side and similar symptoms on the opposite side. Dense strictures were present on both sides. Another patient (Case 35) soon returned and her kidney pelvis reached a capacity of 32 c. c. After the extraction of abscessed teeth the stricture responded to treatment and when last seen several months ago, she was free from kidney symptoms and the pelvis held 10 c. c.

In certain other cases, even with infection, if the kidney pelvis is not too dilated, we get brilliant results in permanently clearing up the symptoms and the infection through dilatation and lavage. This occurred in 6 of my cases, 2 with bilateral stricture and pyelitis.

Case No.	Duration of symptoms	Pelvis before treatment	Pelvis after treatment
34	11 years.	Bilateral and no dilatation.	Free of pus and bacteria.
41	1 year or less.	30 c.c. Staphylococcus.	Free of pus and cocci. Pelvis content 8 c.c. 6 months later.
43	4 months.	Bilateral. Colon. No dilatation.	Free from pus and bacteria.
47	Pyelitis, one week.	11 c.c. Colon.	Free from pus and bacteria.
49	10 months.	40 c.c. Colon.	Free from pus and bacteria. Pelvis content 30 c.c. two months later.
45	Pyelitis 3d month of pregnancy. 4 days' duration of symptoms.	18 c.c. Rod bacillus.	One dilatation with 5 mm. metal bulb. No further symptoms. Examined one year later, and urine normal.

After presenting the above table in my preliminary report, two patients (Cases 34 and 43) both returned with bilateral colon pyelitis. The first (Case 34) was dismissed as well in January, 1915. She again began to have some frequency and discomfort in August, 1915. In January, 1916, 14 teeth were extracted by Dr. Brun, all having bad areas about the roots. She returned in January, 1917, complaining of bladder symptoms and general malaise as on her first visit. After getting the bladder approximately normal and still finding a good deal of pus in the urine, I investigated both kidneys in April and found bilateral strictures and pyelitis. The right kidney was sterile after 2 treatments with 1:500 silver nitrate solution. The left kidney still showed some infection June 1, but she was practically free from symptoms and her general health had improved markedly.

The other woman (Case 43) was dismissed as well in July, 1915. In January, 1916, she returned during an attack of grippe, fearing that her trouble had returned. Investigation of the kidneys showed negative cultures and the ureters apparently well dilated. In February, 1917, the patient was "run down" and had a "slight cystitis attack." She reported in April, 1917, when bilateral pyelitis was found. The left side cleared after a few treatments, but the right side still shows infection (June 1, 1917).

In other cases with infection and large pelves we may be unable by lavage to rid the patients of infection, probably because of the permanently sacculated pelves, but we may restore them to apparent health by doing away with the obstruction in the ureter, thereby relieving their pain and their toxic symptoms (see Fig. 27).

In the following 2 cases with infection the symptoms were relieved by treatment, but I have been unable to follow the patients with a cystoscopy to learn of the final kidney condition.

In Case 14 there were attacks of pain in the kidney for two years, enlarged inflamed tonsils, and arthritis, a stricture about 3 cm. from the bladder, and a kidney pelvis of 12 c. c. capacity with a staphylococcus infection. After a few dispensary treatments she was lost track of.

In Case 23 there was a stone in the bladder to which all the symptoms were referable. The X-ray picture revealed another small shadow in the left ureteral region. On investigation this shadow was found to be outside the ureter, but the ureter had a stricture in the bladder portion and another about 4 cm. from the bladder. The kidney pelvis held 21 c. c. and was infected with the colon bacillus. After a few treatments the strictures were well dilated and the pelvis reduced to 14 c. c. The patient went to her home in North Carolina before the infection had disappeared, but a recent letter, 4 years after her treatment, states that she is perfectly well.

In another group of 3 cases of bilateral, infected pyelitis, the method has not had a fair trial, because of a failure of the patients to persist with treatments after securing enough dilatation to free them from the severe attacks of pyelitis.

Of my second group of 50 cases many are still being treated. Fifteen have been dismissed as well. Thirty-one are classed improved, some of these being practically well, others but little improved. Three had but 1 preliminary investigation and have not been seen since. One had several painful treatments without apparent improvement and, on being urged to have some bad tonsils removed, she ceased treatments.

Treatment by Operation.—When all the methods of vesical approach fail, we have to consider operative measures. No form of operation should be undertaken until as complete investigation as possible has been made of both sides. Stricture of the ureter being bilateral in 30 per cent of the cases, we cannot afford to take anything for granted in dealing with a case in which symptoms may be confined to one side.

If investigation shows stricture of only 1 ureter, associated with a kidney of little or no functional value, conservatism usually calls for extirpation of the injured or dead kidney. This was done in 6 of my first 50 cases with entirely satisfactory results.

If the stricture is high at the junction of the kidney pelvis with the ureter, we may follow Fenger in doing some form of pyelo-ureteroplasty. Actual stricture at this point is extremely rare, and the valve-like obstruction formed by floating kidney can usually be overcome by mere high fixation of the kidney, as I have found in a number of cases.

If careful examination at the time of operation leads one to suspect an organic narrowing at the pyelo-ureteral junction, a pyelotomy and careful dilatation may be done in addition to the kidney fixation, or if the pelvis is very large, a partial

pyelectomy may be done, care being taken to dilate if the orifice into the ureter is at all narrowed.

In Case 44 with symptoms of 4 years' duration there was a stricture in the left broad ligament region and a kidney content of 360 c. c. of clear urine, the functional test being good and equal on both sides. I resected her kidney pelvis and later examination showed the restored pelvis to hold 7.5 c. c.

If the stricture is lower and about the lumbar or pelvic brim region, it has been recommended to cut through above the stricture and implant into the colon or in the loin region.

If the stricture is low and near the bladder, as a vast majority of these strictures are, it has been the custom to implant the severed healthy end into the colon or bladder. I have never made a colon implantation in these cases, but the results of Coffey's work on dogs [11] and of Charles Mayo's work on the human, with Coffey's method, would indicate that there is a field for this method. Some of these patients have a degree of dilatation of the ureter, which would make the operation easier than on the normal ureter, and some of them have such a wide dilatation of the ureter that the operation would be more difficult and the lowered resistance of the kidney would invite infection.

I have done a bladder implantation in 2 of the first 100 stricture cases with indifferent or questionable results in both. I say "questionable results" because in neither case could I later enter the ureter with a catheter from below. Both patients have been in good health since the implantation, but I suspect in both of these cases that there may have formed stricture at the site of the implantation with gradual destruction of the kidney.

Retrograd Dilatation.—I wish to emphasize a method for handling these cases by operation which I have not seen mentioned in the literature but which I am sure must have been employed by some surgeons and which has probably been described before, *viz.*, the treatment by retrograde dilatation. Certainly every surgeon must follow his ureteral stone extractions by dilatation of the usual area of infiltration about the stone.

The ureter is exposed by the extraperitoneal route; incision is made into its dilated portion above the site of stricture, and increasing sizes of the French gum elastic bougies (Fig. 33d) or metal sounds are passed until the stricture is dilated to a diameter of from 5 to 7 mm. The ureteral incision is then closed with catgut. A wick drain is usually left in the extraperitoneal incision for 48 hours to take care of possible contamination by the escaped urine at the time of operation, or of post-operative leakage and the excessive serum secretion following the extraperitoneal operation.

If the dilatation has not been entirely satisfactory or if there has been much trauma to the ureter, I leave it open or close it incompletely with catgut to favor urine drainage in case of temporary swelling sufficient to close the traumatized stricture area. In such cases 2 or 3 small wicks are dropped to a point near the ureteral incision and left for some days or until there is certainly no urine leakage.

A McBurney incision is suitable for most of these cases, but a semilunar line incision is more useful, for it can be enlarged up or down to suit the exigencies of the case, and through a moderately long semilunar line incision one can easily palpate from kidney pelvis to bladder.

With care one can preserve the intercostal vessels and nerves crossing this incision to the rectus muscle simply by deflecting them and working between them.

I have treated 9 of the first 100 cases by this retrograde dilatation, 6 cases in which it was impossible to dilate from below, 2 cases in which stricture of the ureter was found when stone was being looked for, and 1 case in which ureter stricture had been successfully treated from the vesical approach one year previously, but the stricture again swelled shut sufficiently to cause kidney symptoms in the course of an attack of acute gonorrheal salpingitis.

The results in these 9 cases treated by retrograde dilatation have been perfect in 7, so far as measured by relief of symptoms and ability to easily catheterize later from below. In one case, my first one in which I was looking for stone and failed to find it but found the ureter dilated to a diameter of 1 cm. from the bladder to the dilated kidney pelvis, I found a dense stricture in the bladder area and dilated it with the uterine sound only, which probably made a diameter of only 3 mm. I heard one or two years later that this patient developed symptoms again and had a stone removed from the kidney. I have questioned the accuracy of this report, especially as the patient had the kidney removed at a third operation. It is possible that I overlooked a floating stone temporarily lodged in a kidney calyx, but I think it more likely that the symptoms returned and persisted because the stricture was not sufficiently opened. My X-ray picture had shown a questionable shadow in the kidney region.

My second case with a questionable result after retrograde dilatation was one in which the stricture of the left ureter was a diffuse one, reaching from the bladder to 5 or 6 cm. above. The woman had a stricture of the right ureter also, but no symptoms pointing to this side. A few weeks after retrograde dilatation she again showed signs of kidney obstruction and I failed to catheterize from below. I attempted to make a uretero-vesical implantation above the site of the stricture, but failed to find the ureter in the midst of the scar tissue following the previous operation.

The patient got along fairly well and when she was 3 months pregnant, some 3 or 4 years later, her physician at my suggestion tried to catheterize her ureters, but was unable to enter either kidney. One would suppose that with the increased circulation and pliability of the tissues characteristic of the pregnant state, this would be an ideal opportunity for dilating strictures of particular density.

The cases least suited for retrograde dilatation are those in which previous testing of the capacity of the kidney pelvis and ureter and pyelography have shown an absence of marked

[11] Jour. Amer. Med. Assn., 1911, Feb. 11, 397.

FIG. 1.—Illustrating stricture of right ureter due to gunshot injury. The plate is reversed. Symptoms began 8 years after injury. Note stricture just below the pelvic brim, and the slight dilatation of the ureter after 3 months of rather severe kidney symptoms. Bladder urine normal, no infection. Symptoms entirely relieved by three dilatations.

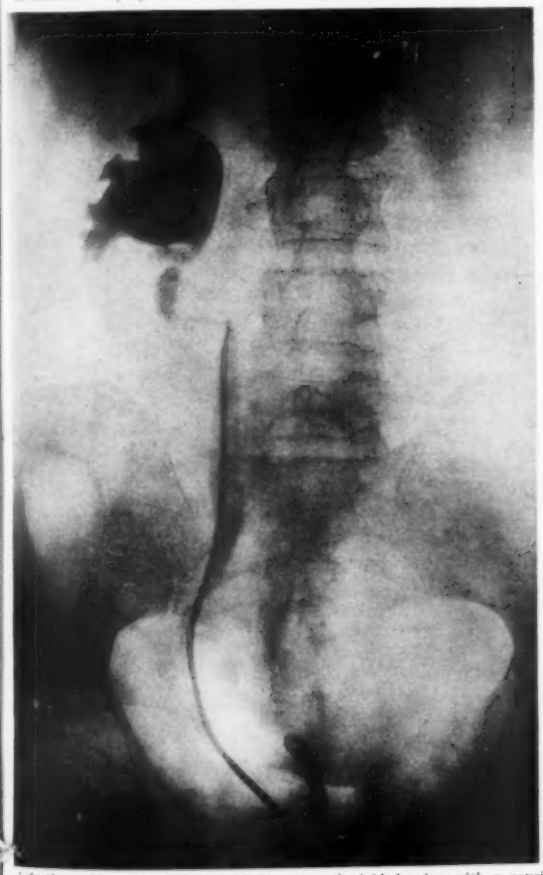

FIG. 2.—Mrs. M., aged 54 years. Referred by Dr. T. B. Johnson of Frederick. Right kidney. Illustrates slight dilatation of pelvic ureter above a stricture in the broad ligament region, and a wider dilatation of the ureter, hydronephrosis, prolapse, and kinks in the upper ureter, due to a stricture in the iliac gland region. X-ray catheter obstructing in a kink. Serious attack of grippe and sinusitis in May, 1916. In June pain and soreness in each hypochondriac region, and somewhat later pain deep in the pelvis on either side. In September first severe attack of pain in the upper right flank which was considered to be either gall-stones or appendicitis, because the urine was reported as normal. In December, 1916, another severe attack of sinusitis and in the following 2 months 3 severe attacks of pain in the upper left flank. When seen February 24, 1917, the patient was having pain in both hypochondriac regions and deep in both sides of the pelvis. Fever 101°, nausea, and some bladder frequency. The urine contained a considerable number of leukocytes, erythrocytes, and a heavy ring of albumin. With this history and these findings a diagnosis was made of bilateral ureteral stricture. Investigation showed multiple strictures on both sides, the left kidney holding 15 c. c. and yielding urine with a few leukocytes but no infection. The right kidney held 33 c. c. and yielded urine with a considerable number of leukocytes, some blood and a colon bacillus infection. The patient showed much improvement after a few dilatations of the strictures during March. She returned home with the idea of coming back for treatments, but in May she was taken with marked symptoms of nephritis, including a purpura and hæmorrhage from both ears. Her physician reported that there was no temperature elevation, a blood pressure of 190-105, and secretion of an almost negligible quantity of phthalein. Death from uremia in June.

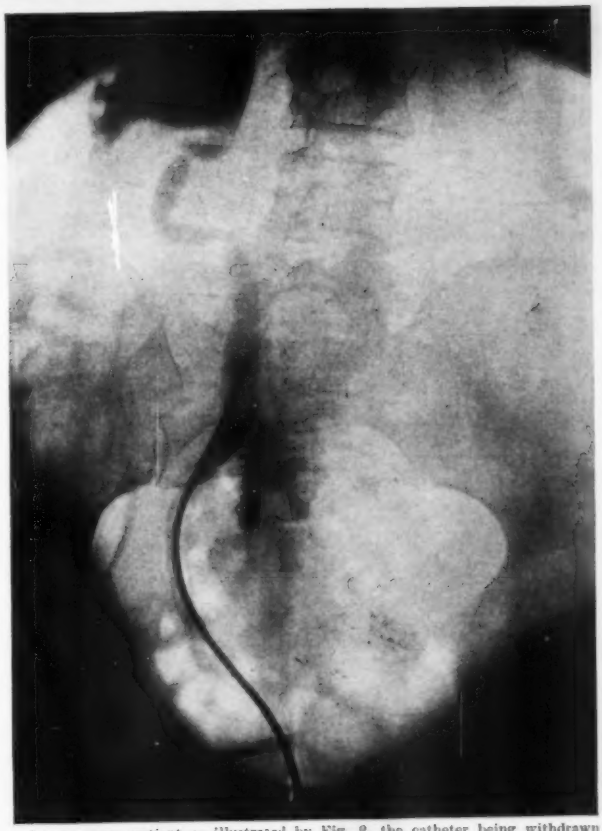

FIG. 3.—Same patient as illustrated by Fig. 2, the catheter being withdrawn until the wax-bulb, located 10 cm. from the tip of the catheter, has obstructed in the upper stricture at the iliac gland region. Note slight dilatation of the ureter between this stricture and the lower stricture in the broad ligament region.

FIG. 4.—Mrs. R., aged 43 years, 2 attacks, December 20, 1915, and February 25, 1916, of symptoms typical of ureter stone, each attack requiring repeated hypodermics of morphin. Great pain beginning in left groin and settling in left kidney region, then extending from kidney to bladder, and causing great frequency of voiding; great distention of bowels, nausea, and vomiting; rectal tenesmus and ineffective attempts at stool. Urine port-wine color, with blood, many leukocytes, negative to culture. Stricture 4 cm. from bladder; kidney holds 16 c. c. Complete relief of symptoms after five dilatations.

FIG. 5.—Patient, 69 years of age, referred by Dr. Hardee Johnston, Birmingham, Ala. Patient admitted to the Church Home and Infirmary May 18, 1917. Since April 5, 1917, four attacks of hæmaturia, each attack lasting four days. No pain, a slight uneasy feeling in the right flank during the attacks. Renal catheter enters right ureter only 3 to 4 cm. Thorium injection beyond catheter of 13 c. c. Later 2 successful dilatations of stricture 3 cm. from the bladder. Report October 1, 1917: no further bleeding and no symptoms.

FIG. 7.—Represents the effect of ureteral stricture on the pelves of a horseshoe kidney. The pict reversed. Mrs. P., aged 55 years, consulted Dr. L. F. Barker in January, 1916, because of attacks o March, 1915, of great abdominal distention, pressure feeling in the back of the neck, and hyster laughing and crying. With these symptoms she had much diarrhea and had lost 40 pounds in weight in year. Careful examinations and analyses resulted in finding a condition of achylia gastrica, and mass in the lower abdomen which Dr. Barker considered a horseshoe kidney. On my first attempt t right ureter could not be catheterized and the left was catheterized with great difficulty becaus strictures near the bladder. After the trauma of this partial failure the patient had a typical attac that night of excessive distention and hysterical noisy wakefulness. Later attempts were successful dilating the ureters, and the patient was greatly improved. The right kidney held 90 c. c. and t left 340 c. c. The urine was negative to culture, the right side being free of abnormal elements a the left containing a few leukocytes. Letter from Dr. C. H. Fair, Greenville, S. C., April, 1 "Patient seems perfectly well."

FIG. 6.—Mrs. H., aged 32 years, treated by Dr. Julius Friedenwald for gastro-intestinal symptoms. Arthritis attacks for past 10 years. Treated in New York 3 years ago for antrum trouble. Pyelitis attacks for past 4 years. Colon bacillus infection left kidney which resists lavage treatment with silver nitrate. On several of the first 6 treatments the catheter was slightly obstructed, but stricture was first suspected because of resistance of the infection to lavage treatment. A large wax-bulb demonstrated stricture 4 cm. above ureter orifice. Kidney pelvis holds less than normal content, but pyeloureterogram shows site of stricture and slightly dilated ureter. Later X-rays showed large dental abscesses. Thorough dilatation of the stricture has resulted in relief from the gastro-intestinal symptoms but the patient still carries a colon bacillus infection of the kidney.

FIG. 8 illustrates a case with almost impassable obstruction from below but in which the characteristi "hang" and scar tissue grating on withdrawal of the wax-bulb were absent. The angulation was du to a fan-shaped band of adhesions suspending the sigmoid bowel to the posterior peritoneum. Thes adhesions probably followed a severe illness with typhoid fever during girlhood.

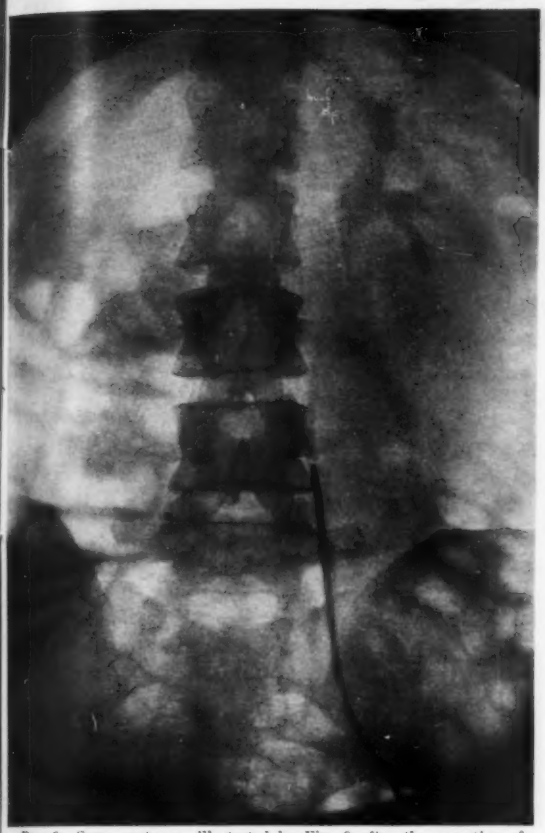

FIG. 9.—Same ureter as illustrated by Fig. 8 after the operation of freeing the sigmoid from the posterior peritoneum.

FIG. 11½.—Case 62. Dense bilateral strictures, ureters slightly dilated, kidney pelves hold only 4 to 5 c. c. Mrs. C., aged 42 years, referred by Dr. Thomas R. Brown because of attacks of pain in the right side like Dietl's crises, accompanied by leukocytes and erythrocytes in the urine. The patient had been operated upon 6 months previously, a suspension of the uterus, appendectomy, and repair of the outlet having been done and she was still complaining of the pain in the right side which had prompted her to seek relief in operation. She described this pain as a constant ache in the right kidney region, extending down the line of the right ureter. When standing or walking the pain was in the posterior flank. When lying down it was more anterior like a "stomach pressure." At all times there was a pain in the ureter region halfway between the kidney and the bony pelvis. Several kidney investigations in May, 1915, failed to discover the source of her trouble. In December, 1915, after becoming interested in ureteral stricture, a wax-bulb was added to the renal catheter and there was a definite hang on its withdrawal. In August, 1916, the patient began complaining of pain in the left side and a dense stricture was found on that side. The dilatations have been carried on irregularly for 2 years and in the meantime the patient has had some abscessed teeth extracted and is now having treatment for infected tonsils and sinuses. She has improved in general health and does not require hypodermics of morphia except on rare occasions after the ureter treatment. Before the discovery of the strictures, she was getting somewhat dependent upon morphia to control the pain.

FIG. 10.—Plate should have been printed to show ureter on right side. Mrs. L., aged 41 years, complained of pain in the lower right pelvis, through the hip and in the thigh since 16 years of age. Considered to be due to a fall across a fence. Also considered as female trouble because the pain came only at the menstrual periods for the first few years. For the last 20 years the pain has come on at any time but is worse at the menstrual period. In the past year more pain in the right kidney region. Operated, J. H. H., March, 1914: Appendectomy and supravaginal hysterectomy for pelvic inflammatory disease and fibroids. Before leaving, the patient told Dr. Watkins, the resident gynecologist, that he had failed to cut out her old pain low in the right side. My associate, Dr. Wynne, made the first examination for ureteral stricture, and the patient said she was sure he had discovered her trouble because she could scarcely use the right hip the day following his treatment. The kidney pelvis held but 5 c. c.; her symptoms were greatly improved after 5 treatments. She will probably need tonsillectomy.

FIG. 11.—Mrs. H., aged 37 years, referred by Dr. L. C. Woodard, Westminster, Md. Pain in the upper right abdomen for 6 or 7 years. For the past few months a gnawing feeling in upper flank, at times in front, at times in back, at times in axillary line. Interfered with getting to sleep. Thinks she feels best when up and moving about. Appendix removed 4 years ago. Recently, X-rays taken in Philadelphia for gall-stones; negative. Gastric analysis = hypo-acidity. Wassermann negative. History of tonsillitis 10 years ago. X-rays of two crowned teeth show abscesses. Urine examination always normal. Test for ureteral stricture, wax-bulb hangs at iliac gland region, and broad ligament region. Pain much relieved after the first examination, and a gain of 5 pounds in four weeks. Only 2 dilatations necessary.

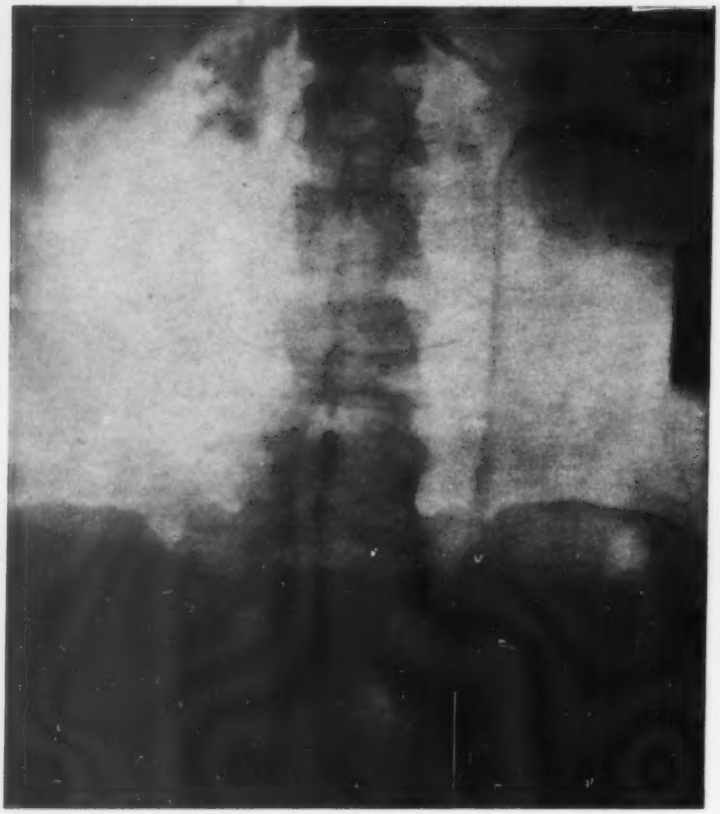

FIG. 12.—Illustrates a patient who had suffered with intermittent renal colic on the left side for 14 years and on the right side for 9 years. The strictures in the broad ligament region were so dense as to be almost impassable, yet the kidney pelves held less than normal and the ureters were but slightly dilated.

FIG. 13.—Same case as Fig. 12, showing slight dilatation of the left ureter.

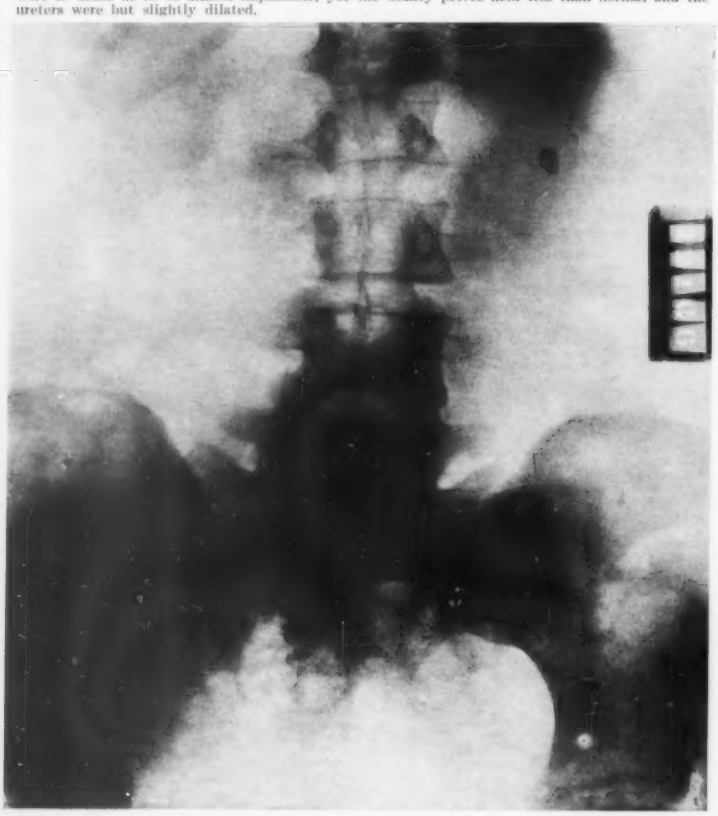

FIG. 14.—Case XVIII, suffering with left kidney colic several years. The X-ray revealed a stone in the pelvis of the kidney. Investigation with the wax-tipped catheter accidentally led to the discovery of ureteral stricture 7 cm. from the bladder, the kidney pelvis holding 8 c. c. Complete relief of symptoms after dilatation of the stricture and the patient refused operation in spite of being shown the X-ray of her kidney stone. This case probably explains the occasional case seen by the surgeon in which pyelotomy for stone is followed by urine drainage for a disappointing length of time, and after healing the patient continues to have symptoms similar to those before complained of.

FIG. 15.—Plate should have been printed to show the shadow of the kidney stone and of the ureter on the right side. Case L. Aged 32 years, the only case in the list of 50 with stricture near the kidney. Catheter diverted laterally just above the pelvic brim and stopped at this same point in radiograms taken on 3 different days. Could not inject sufficient thorium into the kidney to get a definite shadow of pelvis. Nephrectomy; stricture immediately below the enlarged pelvis would not take the fine silver probe. The patient had suffered attacks since early childhood, the pain beginning in the appendix region; and as it grew more severe it traveled diagonally across the abdomen and settled in the left kidney region. Operation three years previously, removing a diseased appendix and chronic pelvic inflammatory trouble, but without influencing her chief symptoms.

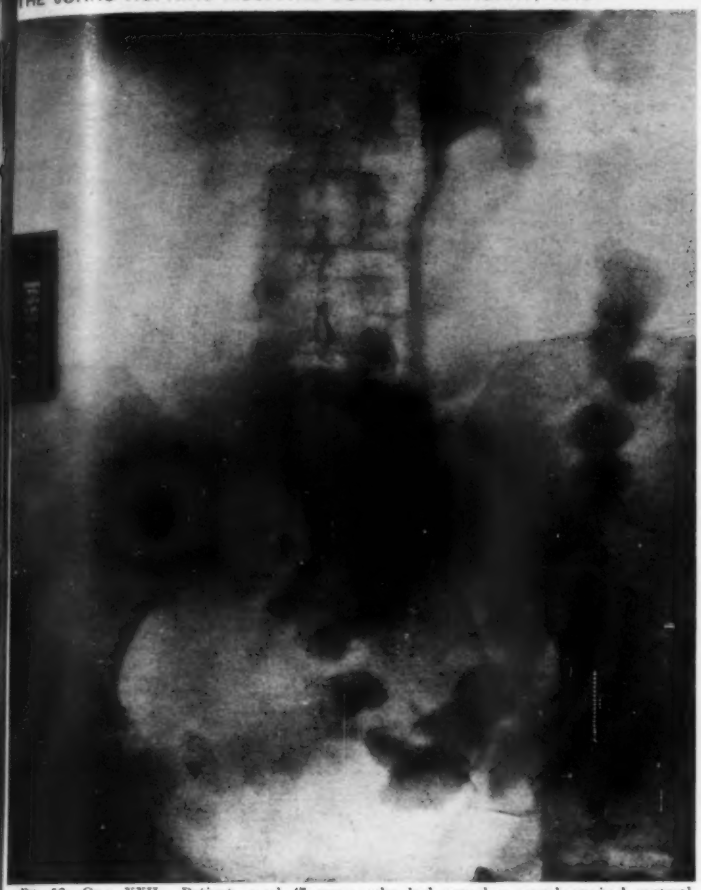

FIG. 16.—Case XXII. Patient, aged 47 years, who had passed a navy-bean-sized ureteral stone 5 years previously. Complained chiefly of pain in the left flank and pressure in the rectum. Failure of catheter to go beyond the pelvic brim region at first examination. Successful catheterization at all later attempts and dilatation with the wax-bulb. Rapid shrinkage of pelvis of the kidney from 28 c. c. to 10 c. c. and relief of symptoms. Observation 5 months later; pelvis of the kidney held 10 c. c.

FIG. 18.—Case XL, aged 26 years, tonsillitis since childhood. Intermittent renal colics on left side for one year, on right side for 3 months. Stone 3 cm. from bladder on left side in dense stricture area. Dense stricture 3 cm. from bladder in right ureter. Bilateral hydronephrosis. Urine not infected. Left ureter exposed by extraperitoneal inguinal incision, stone removed and stricture dilated. Right ureteral stricture dilated later by vesical route.

FIG. 17.—Mrs. C., aged 48 years. Severe renal attacks on right side for 18 months, requiring much morphin. No symptoms on left side. Urine catheterized from bladder shows leukocytes, erythrocytes, colon bacilli, and a trace of albumen. Stricture of right ureter 2 or 3 cm. from bladder. On catheterizing to obtain this thorium X-ray, we found unsuspected obstruction and dilatation of left side. The right side held 38 c. c. and the left 30 c. c. After withdrawing the catheter from the left side and finding scratch marks on the wax-bulb and tip, the small stone shadow was discovered in the lower end of the left ureter, at a point symmetrical with the stricture of the right side. Examination with wax-tip bougie 24 days later showed that the stone had been passed.

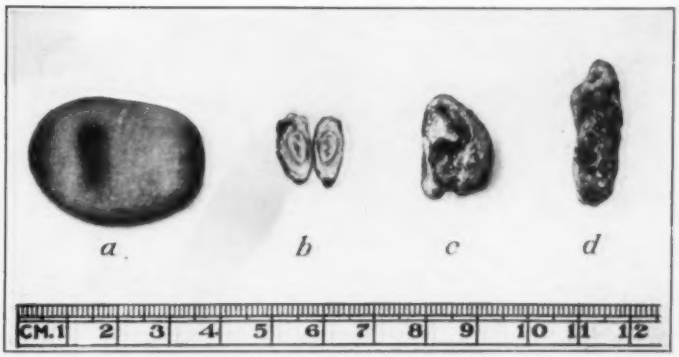

FIG. 19.—*a* gall-stone; *b* appendix stone from author's Case XXXVII (See Fig. 31), whose symptoms were due to ureteral stricture; *c* ureter stone from author's Case XX, showing deep channel for free passage of urine, the patient's hydronephrosis symptoms probably due to a dense ureteral stricture located 3 cm. below the stone and about 3 cm. above the bladder; *d* ureteral stone from author's Case IX, removed from stricture area 5 years after treatment of ureteral stricture.

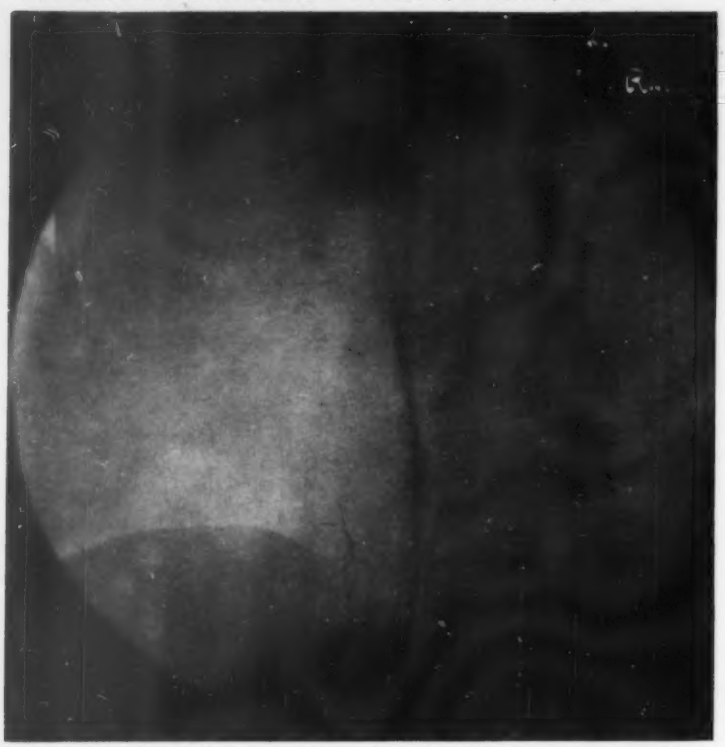

FIG. 20.—Mrs. R., aged 33 years, admitted to J. H. H., Feb., 1917. Four years ago attack of pain in the right side and back with nausea and vomiting. No fever. Was operated upon for appendicitis. Was operated upon one year ago for gall-stones. The doctor said the gall-bladder was shrunken and it was removed. The attacks of pain continued and she has had them once a week or oftener ever since. For the past few months has had these attacks at least once a day. Bearing down pain and burning on urination, 4-5 times at night. Noticed blood in the urine before operation for gall-stones. None since then. Dr. Holmes first catheterized Jan. 22, 1917, and found ureteral stricture, and patient had no more of the daily attacks for the intervening month before my examination. Kidney pelvis 17 c. c. Two points of observation, one 9 cm. and one 3 cm. above the bladder. See Fig. 21.

FIG. 21.—Same patient as illustrated by Fig. 20, showing the X-ray catheter withdrawn until the wax-bulb, 10 cm. from its tip, has obstructed in the upper of the two stricture areas.

FIG. 22.—Mrs. B., aged 28 years, referred by Dr. Bay on May 15, 1917. Attacks of pain in the right side 4-5 years. First attack came suddenly and lasted 2 or 3 hours. Two or 3 attacks within a month, then none for about a year, then awakened one morning at 1 o'clock with such a severe attack she could hardly get out of bed. For the past 3 years 2 or 3 attacks a week, but has gone for 2 or 3 months without an attack. There has been some pain in the side almost every day since the first attack. In Nov., 1916, Dr. Bay did appendectomy and fixation of right kidney. Four days after leaving hospital patient had one of her typical attacks. Kidney holds 15 c. c. Two strictures, one at about the bifurcation of the iliac vessels, and one within 2.5 cm. of the bladder. Symptoms relieved after 2 dilatations.

FIG. 23.—Recent case, ectopic kidney with symptoms due to ureteral strictures. Miss M., aged 60. Pain in the back and gastric symptoms for 15 years. Supravaginal hysterectomy 15 years ago. Persistence of symptoms and 3 years later removal of the cervical stump. Persistence of symptoms and advised to have a fixation of her "floating kidney." Failure to pass the ureter obstruction in the first 3 attempts. Note the catheter arrested at the site of stricture somewhat below the iliac gland region and the thorium injection of an ectopic kidney with its short ureter. Subsequent efforts at dilatation successful. The kidney pelvis held 7 to 8 c. c. Symptoms relieved after 2 dilatations.

FIG. 24.—Case XLIX. Tonsillitis since early childhood. Left hydronephrosis attacks since 2 years of age. Destroyed left kidney removed in 1901, at age of 22 years. Hysterectomy for fibroids January, 1915. Convalescence marked by pyelitis attacks in right kidney. Intermittent lavage for 10 months often followed by severe pyelitis attacks. Pyelogram, Charlottesville, Va.: Right kidney pelvis holds 40 c. c.; ureteral stricture well dilated October, 1915; colon bacillus infection and all symptoms gone December, 1915.

FIG. 26 illustrates the same case as Fig. 25, showing the lower end of the left ureter at the site of stricture.

FIG. 25.—Miss H., Case 70, aged 11 years, referred by Dr. C. A. Woodward, Durham, N. C. For 5 years attacks of acute pyelitis in alternate kidneys accompanied by pain, chills, temperature ranging as high as 105° F., much bladder distress and frequency, and pus in the urine. Tonsillitis since infancy, the tonsils having been removed 1 year before my consultation. The diagnosis of probable bilateral ureteral stricture with secondary colon bacillus pyelitis was made on the foregoing history and examination with the wax-bulb catheter, thorium pyelography, and culture taking confirmed the diagnosis. The illustration demonstrates the difficulty of introducing the proper amount of thorium in a child. It shows the rounded outline of the kidney pelvis with somewhat obliterated calyces and portions of the dilated ureters partially filled with thorium.

FIG. 27.—Patient admitted to The Johns Hopkins Hospital in July, 1912, suffering with repeated chills and a hectic fever ranging as high as 104° F. Right kidney capacity 100 c. c. with a turbid purulent urine. Left kidney capacity 120 c. c. with thick, turbid urine as of a pyonephrosis. Both ureters had infiltrated areas near the bladder, and cultures revealed colon bacillus from either side. After dilating the strictures and washing the kidneys with silver nitrate solution, the patient promptly improved and left the hospital after 3 weeks, feeling well and with urine from each kidney showing only microscopic pus and bacteria. Comparatively good health for four years until death from uremia after this thorium X-ray.

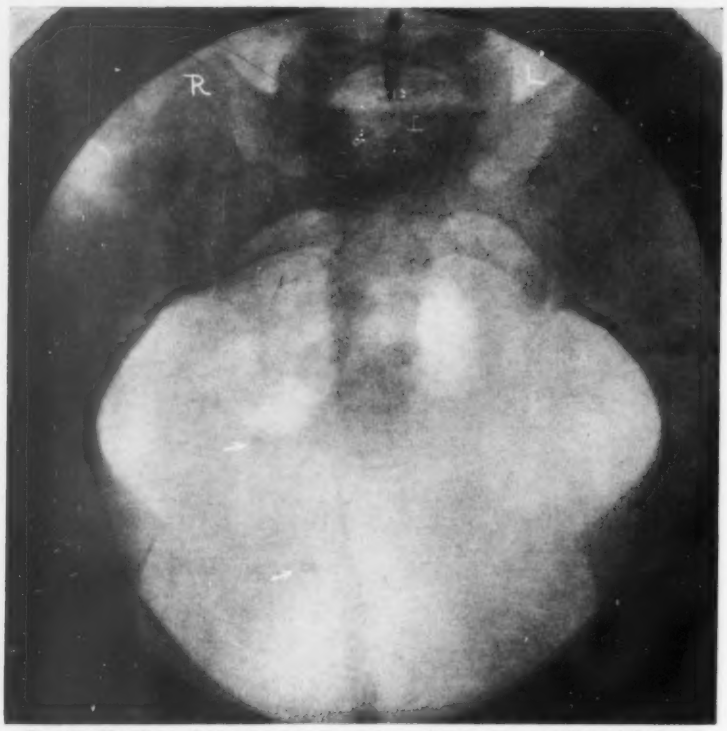

FIG. 28.—Mrs. K., aged 27, referred by Dr. Abrams. Attack of severe pain in right lumbar region radiating down thigh, with high temperature, chills, severe headache, and with burning, strangury and frequency of voiding. This attack 4 months after birth of first child. Breast abscess lanced when baby was 1 month old. Urine, pus and blood and colon bacillus culture. With this X-ray a diagnosis of 2 stones in the lower right ureter. Attempt to get them to pass by dilating with large wax-bulb. Wax tip and bulb negative for scratch marks. Wax-bulb hangs in broad ligament region. Symptoms and colon infection of right kidney cleared after 3 treatments.

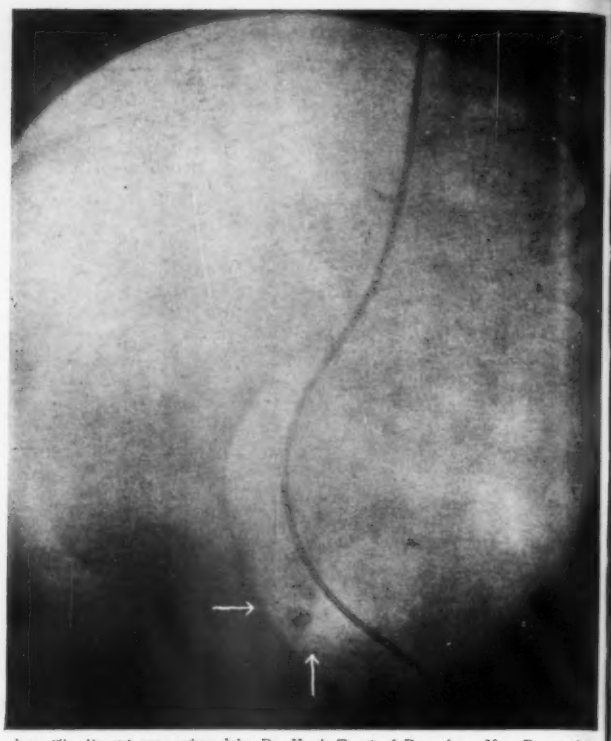

FIG. 29.—Recent case referred by Dr. Hugh Trout of Roanoke. Mrs. R., aged 33, IV-Para, severe pain in right back at intervals for 5 months, 2 of these attacks accompanied by bloody urine. Tonsillitis history for years. Failure of renal catheter to go by an obstruction low in ureter. X-ray showed 3 small shadows apparently in right ureter. Operation failed to demonstrate stone in ureter. Removal of a chronic appendix and of a dense right ovary. A post-operative X-ray demonstrated the same shadows. Persistence of the patient's symptoms. Successful passage of X-ray catheter and demonstration of ureteral stricture and slight hydronephrosis, and obtaining of this picture. Relief of symptoms after 3 dilatations.

FIG. 30.—Recent case referred by Dr. E. L. Mortimer. Mrs. H., aged 20 years, baby 3 months old. Severe attacks for past month, pain alternating in the flanks, temperature as high as 105° F., chills, prostration. Patient had suffered attacks of severe pain in both posterior flank regions since 10 years of age. These attacks were worse after she began to menstruate and were more likely to occur at the time of her period. One surgeon had interpreted them as ovarian attacks. She often had frequency of voiding with the attacks and for this reason and because of their location, they had usually been considered as kidney attacks. She had severe pain in the back all during pregnancy but had not required morphia for any of her colics. Bilateral colon bacillus pyelitis, dense bilateral strictures, kidney pelves both smaller than normal. Bi-weekly dilatation of alternate ureters and lavage with 1: 500 silver nitrate solution for 3 months. Final cessation of all symptoms, negative cultures, restoration to good health.

FIG. 31.—Case 37, aged 57 years. Mrs. K., referred by Dr. Julius Friedenwald: appendix stone, and gall-stone, the acute symptoms due to ureteral stricture. Indigestion for several years. For 2 weeks severe attacks in right side, colicky pains radiating from right lumbar region downward and forward to right groin. Pressure in bladder region, frequency and burning. Chilly feelings and temperature of 103° F. Right kidney enlarged and tender. Pus, blood and bacteria in urine.

Because of questionable position of stone shadow a right semilunar line incision was made. Ureter 1 cm. in diameter traced from bladder to kidney—no stone. Opening made in peritoneum—stone found in base of appendix. Large gall-stone removed through separate incision (see Fig. 19-a). Drainage of gall-bladder—closure of peritoneal incision over appendix region. Incision into ureter—wide dilatation of stricture in bladder wall region. Excellent health since.

FIG. 32.—Mrs. S., aged 29 years, referred by Dr. R. C. Fravel, Richmond. The right kidney had been removed one year previously for calculus. Patient complaining of pain in the left kidney region and low in the left pelvis, and passing thick, purulent urine, yielding colon bacillus culture. Dilatation of stricture 3 cm. above bladder and relief from pain. Repeated lavage failed to clear the urine. X-ray shows eroded character of pelvis outline. Search for tubercle bacilli: present in large numbers.

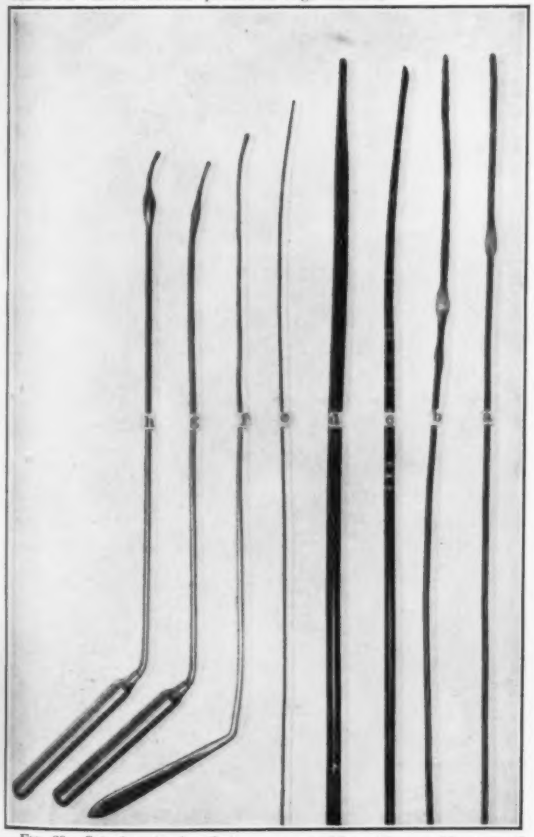

FIG. 33.—Set of ureteral catheters, etc., used by author, *a* blunt round-tip renal catheter with wax-bulb; *b* olive-tip renal catheter with large wax bulb, protected on either side with smaller bulb; *c* Garceau graduated whistle-tip catheter; *d* flexible bougie (7 mm. diam.). Sizes vary from 3 to 10 mm., the smaller sizes being useful for dilating through the cystoscope from below, and the larger sizes for retrograde dilatation from above; *e* whalebone filiform (varying sizes are used through the cystoscope); *f* metal searcher with olive tip; *g* metal bulb dilator 3 mm. with curved olive tip; *h* metal bulb dilator 5 mm. with curved olive tip.

FIG. 34.

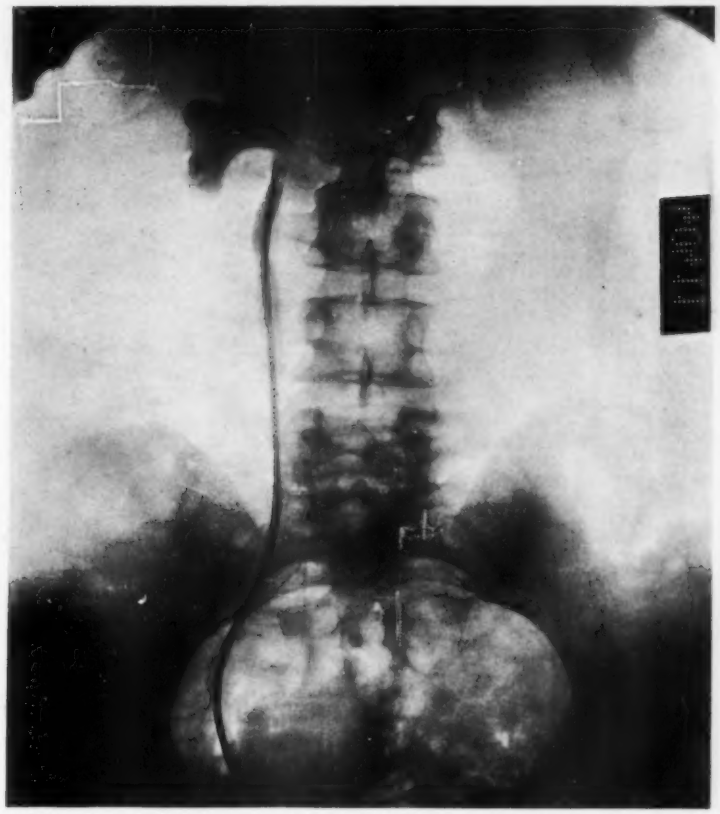

FIG. 35.—Plate should have been made to show the shadow on the left side. Miss H., aged 40 years. Right nephrectomy for stone April, 1900. Consulted Dr. Kelly, May, 1914, for a constant pain in the left kidney region. Investigation of kidney and failure to find cause of the pain. Consulted author in June, 1914, and again in March, 1915, and no cause found in the urinary tract for the pain. Operation, J. H. H., March, 1915: hysterectomy for fibroids and left oöphorectomy for "tender left ovary." Returned in October, 1915, with the same pain in the left kidney region, and examination showed definite stricture and a hydronephrosis of 18 c. c. This hydronephrosis increased to 32 c. c., while the patient was having repeated dilatations of the stricture. After removal of several abscessed teeth the dilatations gave permanent relief, with a shrinkage of the pelvis of the kidney to 10 c. c.

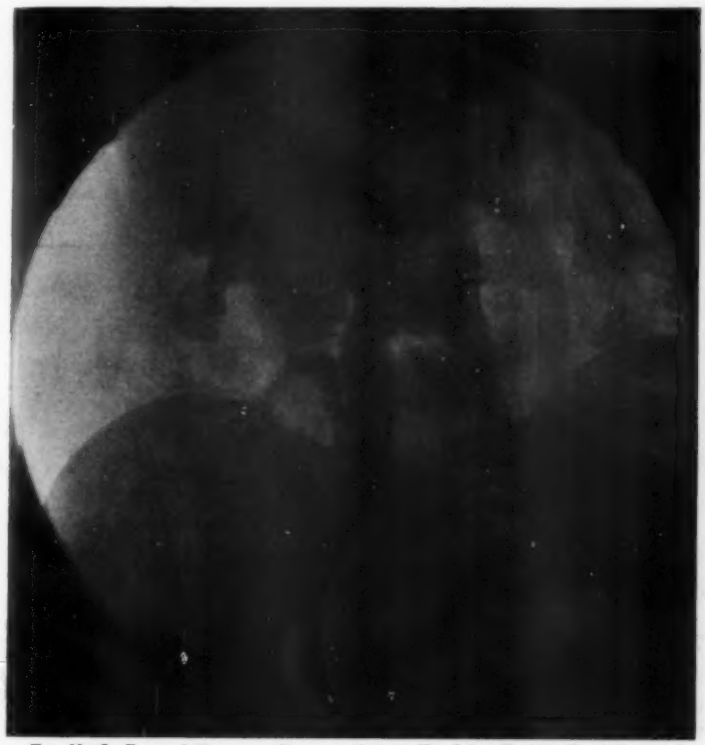

FIG. 36.—L. F., aged 11 years. Recent patient at The Johns Hopkins Hospital. At 7 years of age the left kidney was removed for intermittent hydronephrosis attacks. These attacks began at 3 years of age soon after diphtheria. The attacks came on suddenly and lasted from one hour to several days and then stopped suddenly. There was no history of chills or hematuria. After the nephrectomy the patient was free from attacks for 2 years and at 9 years of age, similar attacks began on the right side and continued for 2 years until we dilated the ureteral stricture in the broad ligament region. There were no attacks after the first dilatation. The patient was dismissed after 3 treatments. There had been throat trouble since early childhood. Enlarged, diseased tonsils were removed while the patient was under treatment for the stricture.

enlargement of the lumen above the site of stricture. In these cases the ureter is found to be too small above the stricture to admit large dilators, and if it is at all possible to get by from the vesical approach, one should be satisfied to do as well as possible by this route although it may require a long, tedious course of treatment.

I cannot speak with authority concerning ureteral stricture work in the male. Because of the restriction in the use of instruments, it is probable that retrograde dilatation will be resorted to much more frequently in the male than in the female. Dr. Bransford Lewis has devised a clever flexible metal dilator which will undoubtedly be of use in some cases. In many cases this instrument will be found to be too blunt to get by the stricture area. Those who use male instruments that take a No. 8 or No. 9 renal catheter will be able to relieve many cases merely by passing the catheter, particularly if the dilatation can be repeated every few weeks or months. In the past, many patients suffering from hydronephrosis and from pyelitis due to stricture have been improved or cured of their symptoms without discovery of the stricture because the passage of a renal catheter gives sufficient dilatation in some cases to result in good drainage.

THE CHIEF FUNCTION OF THE OBLIQUE MUSCLES OF THE EYE

SOME OBSERVATIONS WHICH SEEM TO LEND SUPPORT TO A LONG DISCARDED THEORY

By SAMUEL THEOBALD, M. D.,

Clinical Professor of Ophthalmology, Johns Hopkins University, Medical Department; Ophthalmic Surgeon, Johns Hopkins Hospital and Baltimore Eye, Ear and Throat Hospital, Baltimore

In endeavoring to find an answer to the question, What is the chief function of the oblique muscles of the eye?, it is important that we should take into account the fact that these muscles are not peculiar to man, but that, on the contrary, they are found, almost without exception, in all vertebrates—the only exception being the myxinoids or lampreys. This fact, which shows that they were evolved many thousands of years before even the anthropoids made their appearance, seems to indicate that they have a much more important function to serve than that which is commonly attributed to them in man—that their chief office is to counteract the faulty action of the superior and inferior rectus muscles, owing to the fact that the direction of the pull of these muscles does not correspond with the sagittal axis of the eyeball. Surely these conditions do not exist generally in vertebrates, and, therefore, could have had no bearing on the evolution of the oblique muscles.

In the early part of the last century, the commonly accepted view, which, according to Prof. Joseph Pancoast, dates back to the time of Boerhaave, was that the oblique muscles were the antagonists of the recti and, especially, that they prevented the latter, when acting, from retracting the eyeball into the orbit. This view, however, was not without its opponent, and the first instance I have found of its rejection occurs in an article by Green,[1] in which the author says:

To the favorite doctrine of most anatomical writers, we cannot subscribe, viz., that the obliqui are the *antagonists* of the recti. This error is based on another, viz., that there is a *necessity* for antagonism, *i. e.*, that the recti *retract* the ball from its ordinary position in the socket.

It is to be regretted that, at least in the published abstract of his paper, the writer adduces no facts or observations to sustain these very positive assertions.

Coming down, now, to a more recent time, we find that so careful an observer as Fuchs held that some provision *is* necessary to prevent the eyeball, when performing its movements, from leaving its place in the orbit, and, according to his view, it is the bulbo-orbital fascia which provides against this misadventure.[2] He says:

By means of this system of fasciæ pervading the orbit, the contents of the latter are fixed in place. It is owing to them that the eye does not leave its place when performing its movements, but turns about a fixed center.[3]

This view of the action of the bulbo-orbital fascia, I may add, is held, also, by Duane,[4] another high authority on the ocular muscles and movements, who recently said:

The position of the eye in the orbit seems to me to be governed mainly by the fascial bands (including the check ligaments) that connect the eye and muscles with the orbital walls.

It requires no little temerity on my part to call in question the competence of the bulbo-orbital fascia to do what these authorities agree in holding it does do—to prevent the eye from leaving its place in the orbit, when the recti muscles contract, and to cause it to turn about a fixed point.

[1] Green, C.: On the Functions of the Oblique Muscles of the Eye. Abstr., Boston Med. and Surg. Jour., 1845, xxxii, 191.

[2] Motais, discussing the fixity of the eyeball in the orbit, in his treatise on the "Anatomie de l'appareil moteur de l'œil de l'homme et des vertébrés" (1887, pp. 123-124), answers the question, How can an organ with such yielding walls, immersed in a soft mass, and subject to such rapid movements, maintain a fixed position?, by saying that it is due to the combined action of several anatomic elements, which he mentions in this order: the antagonism of the rectus and oblique muscles, the bulbo-orbital fascia, with its ligamentous wings, and the cushion of adipose tissue which occupies the deeper portion of the orbit.

[3] Fuchs: Text-Book of Ophthalmology, fourth American edition, p. 689.

[4] Personal communication to the author.

To effect this end, it would seem there must be tense, inelastic bands of fascia connecting the sclera or Tenon's capsule with points on the orbital walls anterior to their attachments to the eyeball. That these bands must be, as I have described them, *tense* and *inelastic*, appears obvious; otherwise, having no power of contraction, they could not effectually oppose the very real backward pull of the several recti muscles.

Do such fascial bands exist? And, if they did, would they not, acting as check ligaments, necessarily interfere with the movements of the eye?

These questions, it seems to me, admit of but one answer: No such bands do exist, and, if they did, the rotational movements of the eyeball would certainly be seriously curtailed.

Accepting the view of Fuchs, which I understand that Duane endorses, that *some* provision is necessary to prevent the eye being pulled back into the orbit by contraction of the rectus muscles, and thus enabled to rotate about a fixed point, it would appear that we must fall back on the discarded theory of the supporting or antagonistic action of the oblique muscles for a solution of the problem.

If this view is correct, it would seem to follow that the oblique muscles must take part in all movements of the eye.

Subjective light sensations seen in left half of visual field by right eye: *a*, produced by lateral rotation of the eye; *b*, by downward rotation; *c*, by upward rotation. The upper light sensation, in each instance, is caused by contraction of the inferior oblique, and the lower by contraction of the superior oblique.

Observations which I have recently made of certain subjective light sensations, manifestly caused by contraction of these muscles, appear to show that such, indeed, is the case, with the possible exception of the conjoint action of the interni in effecting convergence. On awakening in the early morning, while the room is still dark, I have observed, projected somewhat into the upper visual field of each eye and in juxtaposition, always one above the other, two subjective light sensations, one decidedly sharper than the other, on rotating the eyes widely in any direction. On converging the eyes strongly, which I found not so easy to do in the dark, I have thought I obtained similar light sensations, but they were less well defined and more evanescent than those produced by rotating the eyes vertically or laterally.

When the eyes are turned upward, in which movement the inferior oblique should act more energetically than the superior, the brighter and better defined light sensation, as we should expect, is, as shown in the illustration, above, the less defined one, produced by the feebler action of the superior oblique, below. When the eyes are turned downward, the superior oblique being then called into more energetic action, the brighter sensation is below, the feebler one above. When the eyes are rotated laterally, the upper and lower light sensations do not differ appreciably in intensity or in form, indicating, it would seem, that in these movements the obliques act with approximately equal energy; and the same is true of those which, I believe, I observed on converging the eyes. The phosphenes seen in the left half of the visual field are, of course, projected from the right eye, and vice versa.

I may add that the light sensations described rapidly decline in brilliance, and, with the exception of those caused by rotating the eyes upward, are best observed with the lids closed, and, further, that repeated attempts to produce them, by energetically rotating the eyes, presently give rise to an ill-defined aching sensation, most noticeable, perhaps, in the region of the trochlea of the superior oblique.

If further observation should show that these subjective sensations do not occur in convergence of the eyes for a near point, a possible explanation might be that in regarding near objects a certain amount of displacement of the eyes backward and inward, in addition to their inward rotation, may not be harmful, may, indeed, even be helpful, so that conjoint action on the part of the obliques under such circumstances would not be called for.

As to the competence of the oblique muscles to counteract the backward pull of the recti, and to maintain the eyeball in its proper position in the orbit, it would seem that they are fully equal to the task. With their fixed attachments close to the orbital border—that of the inferior oblique actually and that of the superior oblique potentially, because of its trochlea—and passing from these points to grasp the eyeball near its posterior pole, one from above, the other from below, and, moreover, unlike the bulbo-orbital fascia, having the power to contract, so as to oppose with exactness the backward pull of the recti, the oblique muscles appear to be entirely capable of causing the eye to rotate, with precision, about a fixed center, a provision so essential to accurate vision, and, especially, to perfect binocular vision.

If it be objected that to do this the direction of the pull of these muscles would have to be straight forward, it may be pointed out that the pull of the recti, especially the superior and inferior recti, is not straight backward, and that the direction in which the energy of the obliques is exerted seems really to be that best adapted to accomplish the end in view.

That the oblique muscles serve other important ends, especially in preventing the superior and inferior recti from tilting the vertical axis of the eye, and in assisting those muscles to rotate the eyeball upward and downward, is, of course, not to be denied; but that their *chief* function is that which I have set forth seems, to me at least, to admit of but little doubt.

NEW PUBLICATIONS.

The following monograph is for sale by The Johns Hopkins Press, Baltimore, Md.:

Relation of Tonsillar and Nasopharyngeal Infections to General and Systemic Disorders. By S. J. Crowe, S. Shelton Watkins and Alma S. Rothholtz. 63 pages. Price, $1.25.

CLINICAL OBSERVATIONS ON THE HEMOGLOBIN AFTER OPERATION

By GEORGE R. DUNN,

Assistant Resident Surgeon, The Johns Hopkins Hospital,

AND

H. M. N. WYNNE,

Assistant Resident Gynecologist, The Johns Hopkins Hospital

There are but few observations in the literature on the hemoglobin of surgical patients after operation. Grünwald in 1889 published a series of 10 cases, from which he concluded that there is little or no oligochromemia immediately post-operative, even after abundant hemorrhage. His work, however, does not show when or for how long the hemoglobin decreases. The lowest point of the hemoglobin curve is of interest to surgeons as a clinical estimate of the amount of blood lost at operation. We have been unable to find any data on this point in the literature. According to Laker (quoted by Grünwald) immediately after operation there is no change, or very slight change, in the hemoglobin, but after several days in cases in which there has been severe hemorrhage there is also a great reduction of hemoglobin. After hemorrhage the total blood volume is decreased, the quality of the blood being first changed when it becomes thinned owing to absorption of fluids from the intestinal tract (Hoppe-Seyler and Penzoldt, quoted by Grünwald).

Young says that the hemoglobin immediately after hemorrhage shows no especial change, but that after 6 to 12 hours the percentage is lowered, the fluid content of the blood seemingly adjusting itself temporarily within the first 24 hours.

Boothby and Berry have shown that the percentage of hemoglobin and the red blood cells are increased under conditions of work, causing an appreciable amount of perspiration, and that there is no increase without sweating.

In all probability the most important factors determining the hemoglobin curve, except the loss of blood at operation, are the intake of fluids, at or after operation, by infusions or through the mouth or rectum, and the output in sweat, vomitus, urine and feces.

In the series of cases here reported, we have endeavored to show the effect upon the hemoglobin of operations during which there has been slight, moderate or severe hemorrhage.

TECHNIQUE

The Sahli Hemiglobinometer was used for hemoglobin determinations, as it is an instrument particularly adapted to general clinical use.

A uniform technique was observed throughout, and in almost all cases one instrument was used for all the readings on a given case. When two instruments were used on the same case, they were carefully checked against each other and corrections made, when necessary. The series of readings on a given case were made by one observer, or, when two took part in it, their findings were carefully controlled and corrections were made when necessary.

In most cases the exact time of an observation has been noted so that the number of hours between any two readings can be determined easily from the tables.

The blood was obtained from the finger or ear, the same source being always employed for any given patient.

In the accompanying tables the date, time and character of the operation are recorded. The loss of blood at operation is only approximately estimated. We lack quantitative data on the intake and output of fluids during the entire time that the patients have been under observation, but notes have been made of all infusions, liquids given by the rectum and of vomiting after operation. Unless otherwise noted, the patients ran the usual course, being encouraged to take water by the mouth as soon as possible.

All of the gynecological patients were operated upon under anesthesia—nitrous oxid and oxygen, followed by ether administered by the open drop method.

CONCLUSIONS

The hemoglobin readings shortly after operation show very little change when compared with readings made before operation, even in cases of severe hemorrhage. The immediate post-operative reading often shows some increase over the pre-operative reading.

The lowest point of the hemoglobin curve is found usually from 30 to 60 hours after operation (a difference of less than 5 per cent is not considered).

The greatest drop in hemoglobin is usually during the first 24 to 36 hours, and is most rapid when salt solution infusions have been given.

In the series showing only a slight loss of blood at operation, the hemoglobin readings are usually higher during the first 12 hours than those made before operation, and there is very little post-operative decrease at any time.

We wish to thank Dr. Howard A. Kelly for permission to publish these observations.

LITERATURE

Boothby and Berry: The Effect of Work on the Percentage of Hemoglobin and Number of Red Corpuscles in the Blood. Amer. Jour. Physiol., XXXVII, p. 378.

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TABLE I.—HÆMOGLOBIN IN CASES WITH VERY SLIGHT BLOOD LOSS AT OPERATION

Gyn. No.	Female	Race	Age	Character of Operation	Operation Date	Blood Loss at Operation	Hb. and blood counts before Operation	After Operation. Day of Operation	2d Day	3Day	4th Day
21836	"	White.	24	Dilatation and curettage.	2/5/16. 9.05 a. m.	Very Slight.	2/4/16. Hb. 105%. W. B. C. 10,000.	7.15 p. m. Hb. 107%.	7.15 p. m. Hb. 102%.	7.15 p. m. Hb. 103%.	7.30 p. m. Hb. 105%.
21845	"	"	60	Amputation of cervix Vag. fixation of uterus Ant. and post. colporrhaphy.	1/24/16. 10.00 a. m.	Slight.	1/22/16. Hb. 87%. W. B. C. 7,400.	9.45 p. m. Hb. 85%.	9.00 p. m. Hb. 86%.	9.15 p. m. Hb. 95%.	9.15 p. m. Hb. 90%.
21861	"	Black.	28	Ether exam.	2/17/16. a. m.	None.	2/16/16. Hb. 78%. W. B. C. 7,000.	3.30 p. m. Hb. 84%. 7.45 p. m. Hb. 78%.	7.30 p. m. Hb. 72%.	.....	.....
21877	"	"	31	Hysteromyomectomy. Double S. O. Appendectomy.	2/9/16. 11.00 a. m.	Slight.	2/8/16. Hb. 80%. W. B. C. 7,600.	6.45 p. m. Hb. 95%.	8.30 p. m. Hb. 95%.	7.45 p. m. Hb. 80%.	7.45 p. m. Hb. 80%.
21878	"	"	42	Appendectomy.	2/8/16. a. m.	Slight.	2/7/16. Hb. 83%. W. B. C. 15,600.	8.00 p. m. Hb. 85%.	6.45 p. m. Hb. 83%.	8.15 p. m. Hb. 85%.	7.40 p. m. Hb. 83%.
21888	"	White.	25	Suspension of uterus. Appendectomy. D. and C. R. R. V. O.	2/10/16. 9. 30 a. m.	Slight.	2/9/16. Hb. 95%. W. B. C. 5,440.	8.00 p. m. Hb. 101%.	7.30 p. m. Hb. 103%.	7.30 p. m. Hb. 99%.	7.30 p. m. Hb. 93%.
21891	"	"	16	D. and C.	2/29/16. 8.20 a. m.	Very Slight.	2/18/16. Hb. 90%.	1.00 p. m. Hb. 101%. 7.45 p. m. Hb. 95%.	8.00 p. m. Hb. 100%.	7.15 p. m. Hb. 90%.	7.45 p. m. Hb. 93%.
21913	"	Black.	38	Hysteromyomectomy. Appendectomy.	2/23/16. 9.45 a. m.	Slight.	2/22/16. Hb. 40%. W. B. C. 11,800. 2/23/16. 9.00 a. m. Hb. 38%.	11.00 a. m. Hb. 34%. 9.00 p. m. Hb. 44%.	7.15 p. m. Hb. 48%.	8.00 p. m. Hb. 41%.	7.45 p. m. Hb. 43%.
21914	"	"	41	Hysteromyomectomy. Double S. O. Appendectomy.	2/14/16. 9.20 a. m.	Slight.	2/8/16. Hb. 53%. W. B. C. 5,400.	12.00 Noon Hb. 67%. 7.15 p. m. Hb. 68%.	.....	7.30 p. m. Hb. 55%.	8.00 p. m. Hb. 50%.
21933	"	White.	34	Hysterectomy. Double S. O.	2/8/16. 10.45 a. m.	Slight.	.....	8.15 p. m. Hb. 88%.	.....	9.00 p. m. Hb. 85%.	.....
21939	"	"	19	Dilatation of cervix. Appendectomy. Suspension of uterus.	2/29/16. 8.45 a. m.	Slight.	2/28/16. Hb. 93%. W. B. C. 9,920.	1.00 p. m. Hb. 105%. 8.00 p. m. Hb. 90%.	.....	7.00 p. m. Hb. 86%.	8.00 p. m. Hb. 90%.
21940	"	"	31	D. and C.	3/14/16. 8.20 a. m.	Very Slight.	3/12/16. Hb. 86%.	8.30 p. m. Hb. 91%.	8.15 p. m. Hb. 90%.	.....	.....
21841	"	"	32	Dilatation of cervix. Cauterization of cervix.	2/5/16. 9.45 a. m.	None.	2/4/16. 7.15 p. m. Hb. 97%.	7.15 p. m. Hb. 100%.	7.30 p. m. Hb. 100%.	7.30 p. m. Hb. 102%.	7.15 p. m. Hb. 105%.
22002	"	"	36	Double salpingectomy. Ligation of ovarian and internal iliac arteries. Percy cautery.	3/14/15. 2.00 p. m.	Very Slight.	3/13/16. Hb. 80%.	8.30 p. m. Hb. 80%.	8.15 p. m. Hb. 80%.	5.45 p. m. Hb. 75%.	.....
22073	"	"	55	Left S. O.	4/18/16. 12.10 p. m.	Slight.	4/17/16. Hb. 102%. W. B. C. 11,000.	8.00 p. m. Hb. 106%.	8.15 p. m. Hb. 108%.	.....	8.15 p. m. Hb. 102%.
22082	"	"	35	Hysteromyomectomy. Hæmorrhoidectomy.	4/17/16. 11.00 a. m.	Slight.	4/13/16. Hb. 110%. W. B. C. 8,000.	9.00 p. m. Hb. 125%.	7.45 p. m. Hb. 121%.	9.00 p. m. Hb. 125%.	.....
22023	"	"	24	Left Salpingectomy.	3/29/16. 9.30 a. m.	Very Slight.	3/28/16. Hb. 83%. W. B. C. 9,160.	7.30 p. m. Hb. 83%.	.....	8.30 p. m. Hb. 82%.	.....
21814	"	Black.	21	Double salpingectomy. Appendectomy.	1/13/16. 10.00 a. m.	Very Slight.	1/12/16. Hb. 69%. W. B. C. 8,000.	.....	Noon. Hb. 65%.	7.00 p. m. Hb. 70%.	7.00 p. m. Hb. 72%. W. B. C. 10,000
21831	"	White.	18	Appendectomy.	1/22/16. 9.30 a. m.	Very Slight.	1/20/16. Hb. 83%. W. B. C. 10,120.	7.00 p. m. Hb. 94%.	7.30 p. m. Hb. 95%.	7.30 p. m. Hb. 95%.	7.15 p. m. Hb. 96%.
21852	"	Black.	34	Hysteromyomectomy.	1/20/16. 8.50 a. m.	Slight.	1/19/16. Hb. 74%. W. B. C. 4,000.	8.00 p. m. Hb. 77%.	7.00 p. m. Hb. 78%.	7.00 p. m. Hb. 75%.	8.00 p. m. Hb. 74%.
21947	"	"	34	Hysteromyomectomy. Appendectomy.	1/15/16. 9.00 a. m.	Slight.	1/13/16. Hb. 75%. W. B. C. 15,600.	7.00 p. m. Hb. 85%.	7.00 p. m. Hb. 77%.	7.00 p. m. Hb. 72%.	8.15 p. m. Hb. 69%.
21974	"	"	36	Hysteromyomectomy. Double salpingectomy. Left oöphorectomy. Appendectomy.	3/14/16. Noon.	Slight.	3/13/16. Hb. 92%. W. B. C. 13,000.	7.30 p. m. Hb. 88%.	7.45 p. m. Hb. 70%.	5.30 p. m. Hb. 83%.	.....
22031	"	White.	47	Hysteromyomectomy. Double S. O. Appendectomy. R. R. V. O.	3/27/16. Noon.	Slight.	2/15/16. Hb. 40%. R. B. C. 2,352,000. W. B. C. 1,600. 3/22/16. Hb. 50%. R. B. C. 3,800,000. W. B. C. 1,100. 3/26/16. 2 p.m. Hb. 54%. 3/27/16. Noon. Hb. 53%.	1.30 p. m. Hb. 64%. 8.45 p. m. Hb. 58%.	.....	9.00 p. m. Hb. 53%.	.....

TABLE I.—HÆMOGLOBIN IN CASES WITH VERY SLIGHT BLOOD LOSS AT OPERATION

5th Day	6th Day	7th Day	8th Day	9th Day	10th Day			Fluids Taken. Vomiting	Remarks
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....	2/6/16. 11.00 a. m. Hb. 91%.	.....	Water well taken after operation. No vomiting.	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
.....	.....	7.45 p. m. Hb. 82%.	.....	.....	.....	2/24/16. 7.45 p. m. Hb. 75%.	.....	.....	.....
.....	.....	.....	8.00 p. m. Hb. 82%.	.....	.....	2/24/16. 7.30 p. m. Hb. 76%.	.....	No vomiting.	.....
8.00 p. m. Hb. 86%.	7.15 p. m. Hb. 89%.	.....	.....	.....	8.15 p. m. Hb. 87%.	2/27/16. 7.45 p. m. Hb. 92%.	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	Water well taken after operation.	.....
7.15 p. m. Hb. 46%.	.....	.....	7.15 p. m. Hb. 43%.	.....	7.15 p. m. Hb. 50%.	3/7/16. 7.45 p. m. Hb. 48%.	.....	Water not well taken after operation.	.....
7.45 p. m. Hb. 55%.	.....	.....	.....	7.45 p. m. Hb. 58%.	.....	2/27/16. 7.30 p. m. Hb. 61%.	3/10/16 10.30 a. m. Hb. 68%.	.....	.....
.....	6.00 p. m. Hb. 78%.	.....	.....	.....	Hb. 88%. W. B. C. 22,000	2/26/16. 8.30 p. m. Hb. 83%.	.....	Water not well taken after operation. Slight vomiting.	Post-op. pelvic cellulitis.
.....	8.30 p. m. Hb. 90%.	.....	7.30 p. m. Hb. 91%.	.....	.....	.....	.....	Water well taken after operation.	.....
7.30 p. m. Hb. 90%.	.....	.....	.....	.....	.....	.....	.....	.....	Slight post-op. bleeding.
.....	.....	.....	.....	.....	.....	.....	.....	Some vomiting.	.....
7.45 p. m. Hb. 75%.	.....	.....	7.45 p. m. Hb. 77%.	.....	.....	.....	.....	Water well taken after operation.	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
8.00 p. m. Hb. 112%.	.....	.....	.....	.....	.....	5/5/16. 8.00 p. m. Hb. 112%. R. B. C. 6,120,000.	.....	.....	.....
.....	.....	.....	8.45 p. m. Hb. 82%.	.....	.....	.....	.....	.....	.....
7.00 p. m. Hb. 71%.	.....	.....	.....	.....	.....	1/28/16. Noon. Hb. 79%.	.....	.....	.....
.....	.....	Noon. Hb. 95%.	.....	.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	Noon. Hb. 77%.	.....	.....	.....	.....	.....
.....	7.00 p. m. Hb. 68%.	.....	.....	.....	.....	1/28/16. Noon. Hb. 89%.	.....	.....	.....
7.30 p. m. Hb. 78%.	.....	.....	8.00 p. m. Hb. 77%.	.....	.....	.....	.....	.....	.....
8.15 p. m. Hb. 55%.	.....	.....	.....	.....	8.15 p. m. Hb. 59%.	4/10/16. 7.30 p. m. Hb. 60%.	4/17/16. 9.00 p. m. Hb. 61%.	.....	.....

TABLE II.—HÆMOGLOBIN IN CASES WITH MODERATE BLOOD LOSS

Gyn. No.	Female	Race	Age	Operation	Date	Blood Loss of Operation	Before Operation	After Operation. Day of Operation	2d Day	3d Day	4th Day
21647	"	White.	39	Curettage and cauterization of cervix.	11/2/15. 9.45 a. m.	Moderate.	11/1/15. Hb. 62%.	.....	Noon. Hb. 57%.	Noon. Hb. 54%.	.....
21799	"	"	14	D. and C.	1/20/16. 8.20 a. m.	Moderate.	1/19/16. Hb. 35%.	.....	Noon. Hb. 30%.	Hb. 31%.	7.45 p. m. Hb. 28%.
22615	"	"	39	Dilatation of cervix and manual removal of placenta.	11/25/16. 8.45 p. m.	Very slight. See "Remarks."	11/25/16. 8.30 p. m. Hb. 61%. W. B. C. 15,500. R. B. C. 3,880,000.	.....	7.45 p. m. Hb. 53%. R. B. C. 3,600,000.	.....	7.45 p. m. Hb. 52%. W. B. C. 9,200. R. B. C. 3,900,000
21875	"	"	30	D. and C. R. R. V. O.	2/8/16. 8.20 a. m.	Moderate.	2/7/16. Hb. 90%. W. B. C. 17,520.	7.45 p. m. Hb. 94%.	7.00 p. m. Hb. 85%.	8.00 p. m. Hb. 82%.	7.15 p. m. Hb. 82%.
21879	"	"	19	Hysterectomy (bi-section). Double salpingectomy. Right oöphorectomy.	2/10/16. 11.55 p. m.	Moderate.	2/9/16. Hb. 93%. W. B. C. 8,200.	8.45 p. m. Hb. 86%.	8.00 p. m. Hb. 83%.	7.45 p. m. Hb. 75%.	8.00 p. m. Hb. 70%.
21931	"	"	36	Hysteromyomectomy. Double salpingectomy. Appendectomy. Anterior and posterior colporrhaphy.	2/26/16. 11.25 a. m.	Moderate.	2/25/16. Hb. 57%. W. B. C. 9,400.	2.00 p. m. Hb. 62%. ½ hr. after op. 7.45 p. m. Hb. 57%.	7.00 p. m. Hb. 54%.	8.00 p. m. Hb. 50%.	7.15 p. m. Hb. 46%.
21993	"	"	33	Hysteromyomectomy. Rt. salpingectomy. Appendectomy.	2/29/16. a. m.	Moderate.	2/28/16. Hb. 86%. W. B. C. 8,000.	1.50 p. m. Hb. 70%. 7.30 p. m. Hb. 80%.	7.45 p. m. Hb. 84%.	6.45 p. m. Hb. 77%.	7.00 p. m. Hb. 70%.
22046	"	"	47	Amputation cervix. R. R. V. O. Cauterization fissure in ano.	3/14/16. 8.45 a. m.	Moderate.	3/13/16. Hb. 86%. W. B. C. 5,360.	8.00 p. m. Hb. 70%.	8.00 p. m. Hb. 70%.	6.00 p. m. Hb. 72%.	.....
22128	"	"	23	D. and C. Posterior vaginal celiotomy. Resection of left ovary.	5/13/16. 10.20 a. m.	Slight. See "Remarks."	5/13/16. 10.20 a. m. Hb. 100%. W. B. C. 9,500.	8.00 p. m. Hb. 100%.	8.00 p. m. Hb. 80%.	.....	9.00 p. m. Hb. 88%.
20883	"	"	20	Panhysterectomy. Double salpingectomy. Left oöphorectomy.	1/25/15. a. m.	Slight. 1/25/15. Hæmorrhage on ward.	Not made.	2/6/15. 8.00 p. m. Hb. 65%. 6.00 p. m. Severe bleeding.	8.00 p. m. Hb. 38%. Very little bleeding.	Hb. 35%. No bleeding.	Hb. 39%. No bleeding.
20900	"	"	21	D. and C. cauterization cervix. R. R. V. O. Hæmorrhoidectomy.	1/28/15. a. m.	Slight. 2/6/15. 6.00 p. m. Severe hæmorrhage.	Not made.	2/6/15. 8.30 p. m. Hb. 80%. 10.30 p. m. Hb. 70%.	Noon. Hb. 70%. No bleeding.	10.00 a. m. Hb. 55%. No bleeding.	Hb. 53%. No bleeding.

TABLE II.—HÆMOGLOBIN IN CASES WITH MODERATE BLOOD LOSS

5th Day	6th Day	7th Day	8th Day	9th Day	10th Day					Fluid Taken. Vomiting	Remarks
Noon. Hb. 48%.	8.30 p. m. Hb. 50%.	.....	.....	8.30 p. m. Hb. 54%.	.....	11/12/15. Hb. 58%.	.....	.....	.....	.....	.....
7.15 p. m. Hb. 34%. R. B. C. 3,864,000.	.....	.....	Hb. 42%.	.....	.....	.....	.....	.....	.....	.....	Pt. had constant bleeding for 2 mos. due to endometrial hyperplasia.
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	Water well taken after operation. No vomiting.	Began to bleed 2 days before op. Profuse hæmorrhage 5 hrs. before op.
7.15 p. m. Hb. 77%.	7.30 p. m. Hb. 75%.	8.00 p. m. Hb. 73%.	7.00 p. m. Hb. 71%.	.....	.....	2/19/16. 8.15 p. m. Hb. 80%.	.....	.....	.....	Considerable vomiting day after operation.	.....
7.15 p. m. Hb. 73%.	.....	7.45 p. m. Hb. 74%.	.....	.....	.....	2/22/16. 7.45 p. m. Hb. 71%.	.....	.....	.....	.....	.....
7.30 p. m. Hb. 46%.	.....	7.30 p. m. Hb. 50%.	.....	.....	.....	3/7/16. 7.30 p. m. Hb. 54%.	3/25/16. 7.30 p. m. Hb. 52%.	.....	.....	Salt sol. 300 c. c. p. r. q. 4 h. day of operation. No vomiting.	.....
.....	8.30 p. m. Hb. 80%.	.....	8.00 p. m. Hb. 78%.	.....	.....	3/15/15. 7.45 p. m. Hb. 80%.	.....	.....	.....	.....	.....
8.00 p. m. Hb. 69%. W.B.C.7,800.	.....	.....	8.00 p. m. Hb. 70%.	.....	.....	.....	.....	.....	.....	Some vomiting for 2 days. Post op.	.....
8.45 p. m. Hb. 88%.	.....	7.45 p. m. Hb. 90%.	.....	.....	.....	.....	.....	.....	.....	Very little vomiting. Post op.	Pt. says she had hæmorrhage night of 5/12/16 and morning of 5/13/16.
10.00 a. m. Severe bleeding. 8.00 p. m. Hb. 41%.	8.00 p. m. Hb. 30%. No bleeding.	4.00 p. m. Hb. 20%. R. B. C. 1,200,000. No bleed'g.	7.00 p. m. Hb. 24%. No bleeding.	.....	2/16/15. 5.00 p. m. Hb. 30%. No bleeding.	2/19/15. 4.00 p. m. Hb. 22%. No bleeding.	2/23/15. Hb. 12%. 2/20/15. Hæmorrhage.	2/26/15. Hb. 18%. No bleeding.	2/28/15. Hb. 25%. 3/2/15. Hb. 35%. No bleed'g.	2/12/15. Indirect transfusion 300 c. c. 2/22/15. Salt sol. infusion.	Hæmorrhage was from a small vessel in vaginal vault.
Hb. 46%. No bleeding.	Hb. 50%. No bleeding.	.....	.....	Profuse hæmorrhage.	2/19/15. Hb. 64%.	2/21/15. Hb. 68%.	.....	.....	.....	2/8/15. Salt sol. infusion 600 c. c.	Bleeding began 6/4/15. Not severe until 6/6/15 at 6.00 p.m.

TABLE III.—HÆMOGLOBIN IN CASES OF SEVERE HÆMORRHAGE

Gyn. No.	Female	Race	Age	Operation	Date	Blood Loss at Operation	Before Operation	After Operation. Day of Operation	2d Day	3d Day	4th Day	5th Day
10170	"	White.	27	Dilatation and curettage. Rt. S. O.	1/7/03.	.....	1/6/03. Hb. 40%.	.....	Hb. 30%.	.....	Hb. 37%.	.....
10698	"	"	25	Rt. S.	8/23/03.	.....	8/23/03. Hb. 15%.	.....	.....	Hb. 10%.	.....	.....
20777	"	"	24	Resection of rt. uterine cornu.	11/30/14. 1.30 a. m.	.....	11/30/14. 1 a. m. Hb. 70%. W. B. C. 20,000.	3.00 p. m. Hb. 50%. W. B. C. 12,000.	Hb. 34%.	Hb. 30%.	Hb. 40%.	.....
22658	"	Black.	21	Resection of left tube.	12/2/16. 10.00 p. m.	.....	12/2/16. 9 p. m. Hb. 56%. W. B. C. 10,600. R. B. C. 3,950,000.	11.00 p. m. Hb. 50%. W. B. S. 20,000. R. B. C. 4,200,000.	11.30 a. m. Hb. 40%. R. B. C. 2,440,000.	12.30 p. m. Hb. 30%. R. B. C. 1,936,000.	12.30 p. m. Hb. 27%. R. B. C. 2,120,000.	7.30 p. m. Hb. 24%. R. B. C. 1,960,000.
22239	"	"	24	Pelvic puncture. Rt. salpingectomy.	6/22/16. 10.00 p. m.	.....	6/19/16. Hb. 82%. W. B. C. 9,100.	10.30 a. m. Hb. 70%. W. B. C. 7,600. R. B. C. 4,352,000. 9.30 p. m. Hb. 70%.	8.30 p. m. Hb. 95%.	9.00 p. m. Hb. 49%. R. B. C. 3,268,000.	9.30 p. m. Hb. 50%.	9.30 p. m. Hb. 49%.
21760	"	White.	34	Double sapingectomy.	12/18/15. 11.00 a. m.	.....	12/17/15. Hb. 61%. W. B. C. 13,600.	Noon. Hb. 61%.	4.00 p. m. Hb. 50%.	8.00 p. m. Hb. 46%.	.....	5.00 p. m. Hb. 50%.
21679	"	"	37	Hysteromyomectomy. Double S. O.	11/15/15. 11.00 a. m.	Severe hæmorrhage.	Not made.	7.45 p. m. Hb. 130%.	9.30 p. m. Hb. 122%.	7.00 p. m. Hb. 105%.	8.00 p. m. Hb. 98%. W. B. C. 9,000. R. B. C. 5,000,000. Differential normal.	8.00 p. m. Hb. 90%.
21893	"	Black.	30	Pelvic puncture. Hysterectomy (bi-section). Double S. O.	2/16/16. Noon.	Severe hæmorrhage.	2/15/16. Hb. 73%. W. B. C. 21,600.	2.30 p. m. ½ hr. after operation. Hb. 83%. 7.30 p. m. Hb. 72%.	8.00 p. m. Hb. 47%.	8.00 p. m. Hb. 45%.	7.45 p. m. Hb. 50%.	.....
21975	"	"	32	Hysteromyomectomy. Double S. O.	3/13/16. 11.25 a. m.	Considerable.	3/11/16. Hb. 80%. W. B. C. 7,200.	1.00 p. m. Hb. 76%. 9.00 p. m. Hb. 68%.	7.30 p. m. Hb. 52%.	7.30 p. m. Hb. 46%.	5.30 p. m. Hb. 47%.	.....
21980	"	White.	40	Panhysterectomy. Left S. O. R. R. V. O.	3/8/16. 11.50 p. m.	Considerable.	3/7/16. Hb. 49%. W. B. C. 12,800.	Hb. 40%.	Hb. 35%.	.....	Hb. 33%.	.....
22024	"	Black.	49	Hysteromyomectomy. Right S. O. Appendectomy.	4/1/6. 12.45 p. m.	Considerable.	3/31/16. Hb. 72%. W. B. C. 7,200.	3.30 p. m. Hb. 64%.	3.00 p. m. Hb. 45%.	4.00 p. m. Hb. 53%.	.....	8.00 p. m. Hb. 50%.
22029	"	"	37	Nephrectomy, Left. Hæmorrhoidectomy.	2/16/16. 10.50 a. m.	Considerable.	2/1/16. Hb. 40%. W. B. C. 5,200. R. B. C. 2,160,000. 2/15/16. Hb. 40%. W. B. C. 1,600. R. B. C. 2,352,000.	Noon. Hb. 38%. 7.30 p. m. Hb. 28%.	7.45 p. m. Hb. 26%.	3.00 p. m. Hb. 25%. W. B. C. 2,320.	8.00 p. m. Hb. 24%.	.....
22138	"	White.	57	Cauterization of cervix with high heat.	5/23/16. 11.40 a. m.	Considerable.	5/22/16. Hb. 49%. W. B. C. 14,200.	2.00 p. m. Hb. 45%. 7.30 p. m. Hb. 38%.	7.15 p. m. Hb. 31%.	7.15 p. m. Hb. 30%.	.....	.....
22180	"	"	33	Pelvic puncture. Exploratory laparotomy.	5/17/16. 12.30 p. m.	Considerable.	5/15/16. Hb. 95%. W. B. C. 19,000. 5/16/16. 5 p. m. Hb. 96%.	2.00 p. m. Hb. 95%. 9.00 p. m. Hb. 92%.	7.00 p. m. Hb. 81%.	8.00 p. m. Hb. 70%.	7.30 p. m. Hb. 68%.	8.30 p. m. Hb. 68%.
22192	"	Black.	32	Panhysterectomy. Double S. O.	5/22/16. 10.30 a. m.	Severe hæmorrhage.	5/21/16. Hb. 70%. W. B. C. 8,920.	1.00 p. m. Hb. 70%. 7.15 p. m. Hb. 60%.	7.30 p. m. Hb. 42%.	7.30 p. m. Hb. 40%.	7.30 p. m. Hb. 41%.	.....
22219	"	White.	31	Removal of retained placenta.	6/12/16. 4.00 a. m.	.....	5/30/16. Hb. 85%. 6/12/16. 4.00 a. m. Hb. 51%.	4.30 a. m. Hb. 51%. 8.00 p. m. Hb. 43%.	8.30 p. m. Hb. 30%.	8.00 p. m. Hb. 27%.	8.00 p. m. Hb. 31%.	.....
22593	"	"	41	Hysteromyomectomy. Double S. O.	11/4/16. 8.40 a. m.	Profuse hæmorrhage.	11/2/16. Hb. 68%. W. B. C. 8,700.	½ hr. after operation. Hb. 75%. R. B. C. 4,240,000.	4.30 p. m. Hb. 55%. W. B. C. 11,600. R. B. C. 3,400,000.	7.00 p. m. Hb. 36%.	11.00 a. m. Hb. 39%. R. B. C. 2,432.000.	.....
21160	"	"	39	Panhysterectomy. Double S. O.	5/6/15. a. m.	Profuse hæmorrhage.	5/5/15. Hb. 80%. W. B. C. 6,700.	.....	Hb. 70%. W. B. C. 13,600.	.....	Hb. 66%. W. B. C. 8000.	Hb. 54%.
21321	"	"	33	Supravaginal hysterectomy.	6/30/15. 11.00 a. m.	Slight. See "Remarks"	6/29/15. 2.00 p. m. Hb. 63%. 7.15 p. m. Hb. 45%. 6/30/15. 10.45 a. m. Hb. 47%.	.....	Hb. 34%.	Hb. 31%.	Hb. 31%.	Hb. 31%.

TABLE III.—HÆMOGLOBIN IN CASES OF SEVERE HÆMORRHAGE

6th Day	7th Day	8th Day	9th Day	10th Day						Fluid Taken. Vomiting	Remarks
.....	.....	.....	.....	.....	10/19/03. Hb. 45%.	.....	.....	.....	.....	.....	Ruptured tubal pregnancy. Abd. full of blood at op. Onset 1/5/03.
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	Blood not removed from abd. cavity at operation.	Ruptured tubal pregnancy. Ab. full of blood at op. Onset 8/16/03.
.....	Hb. 45%.	.....	.....	.....	12/10/14. Hb. 55%.	12/13/14. Hb. 50%.	.....	.....	.....	Infusion of salt sol. 1000 c. c. begun at op. Water taken greedily after op. No vomiting.	Ruptured tubal pregnancy. About 2 litres blood in abd. Onset 12 hrs. before op.
.....	7.30 p. m. Hb. 20%. R. B. C. 2,300,000.	.....	.....	.....	12/16/16. 11.00 a. m. Hb. 42%. W. B. C. 8,200 R. B. C. 3,624,000.	.....	.....	.....	.....	Infusion of salt sol. 1500 c. c. begun at op. Water taken well after op. Very little vomiting.	Ruptured tubal pregnancy. Onset about 36 hrs. before op. Abd. filled with fresh blood.
.....	.....	9.15 p. m. Hb. 52%. R. B. C. 3,680,000.	.....	.....	.....	.....	.....	.....	.....	Infusion of salt sol. 2500 c. c. begun at op. 1 litre of salt sol. left in abd. cavity at end of op. No vomiting.	Tubal pregnancy ruptured ½ hr. before op. under ether. About 2 litres of fresh blood in abd. at op.
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	Tubal pregnancy ruptured about 36 hrs. before op. Estimated 600 c. c. blood in abd. at op.
8.45 p. m. Hb. 88%.	5.45 p. m. Hb. 88%.	8.30 p. m. Hb. 95%.	8.15 p. m. Hb. 95%.	.....	11/26/15. 11.45 a. m. Hb. 95%.	.....	.....	.....	.....	Salt sol. infusion 1000 c. c. at op. Water well taken after op.	.....
.....	7.40 p. m. Hb. 58%.	.....	.....	.....	2/27/16. 7.30 p. m. Hb. 58%.	3/3/16. 7.30 p. m. Hb. 60%.	.....	.....	.....	Salt sol. infusion 1900 c. c. begun at op.	.....
7.15 p. m. Hb. 55%.	.....	.....	8.30 p. m. Hb. 53%.	.....	.....	.....	.....	.....	.....	Salt sol. infusion 1200 c. c. begun at op. Salt sol. p. r. 300 c. c. q. 4 h.	.....
Hb. 29%.	.....	Hb. 37%.	.....	Hb. 35%.	3/19/16. Hb. 39%.	3/21/16. Hb. 42%.	3/22/16. Hb. 49%.	3/23/16. Hb. 52%.	3/26/16. Hb. 52%. 3/30/16. Hb. 62%.	.....	.....
.....	7.45 p. m. Hb. 50%.	.....	.....	.....	4/11/16. 5.00 p. m. Hb. 58%.	.....	.....	.....	.....	.....	.....
.....	7.30 p. m. Hb. 20%.	8.00 p. m. Hb. 25%. R. B. C. 1,368,000.	.....	.....	2/26/16. Hb. 30%. W. B. C. 8,200. R. B. C. 1,228,000.	2/29/16. Hb. 30%.	3/3/16. Hb. 36%.	.....	.....	Salt sol. infusion 3100 c. c. during 24 hrs. after op.	.....
.....	.....	Hb. 37%. W. B. C. 13,000.	.....	.....	.....	.....	.....	.....	.....	Salt sol. 200 c. c. per rectum q. 4 h.	Died 6/1/16 from general peritonitis.
.....	8.00 p. m. Hb. 70%.	.....	.....	.....	6/4/16. Hb. 77%.	.....	.....	.....	.....	.....	.....
.....	.....	7.30 p. m. Hb. 50%.	.....	.....	6/5/16. Hb. 60%.	.....	.....	.....	.....	.....	.....
8.15 p. m. Hb. 33%.	.....	.....	.....	.....	6/23/16. Noon. Hb. 56%.	6/29/16. 8.30 p. m. Hb. 53%.	.....	.....	.....	Salt sol. infusion 2500 c. c. begun at op.	Bleeding began 6/11/16. 3.30 p. m. Severe hæmorrhage 6/12/16 1.00 a. m. pack replaced.
4.30 p. m. Hb. 43%. R. B. C. 3,348,000.	.....	.....	.....	.....	11/14/16. 11.45 a. m. Hb. 46%. W. B. C. 15,800. R. B. C. 3,208,000.	11/24/16. 3.30 p. m. Hb. 51%. R. B. C. 3,648,000.	.....	.....	.....	Salt sol. infusion 840 c. c. on table. Salt sol. p. r. 1200 c. c. 1st 18 hrs. No vomiting.	.....
Hb. 62%.	.....	.....	Hb. 70%.	.....	5/21/15. Hb. 75%.	.....	.....	.....	.....	Salt sol. infusion on table 700 c. c. · Salt sol. p. r. 300 c. c. q. 4 h. onward.	.....
Hb. 36%.	Hb. 34%.	Hb. 37%.	.....	Hb. 40%.	7/12/5. Hb. 41%.	7/15/15. Hb. 42%.	7/16/15. Hb. 48%.	7/18/15. Hb. 42%.	7/27/15. Hb. 50%. 7/19/15. Hb. 42%.	Salt sol. infusion 900 c. c. begun at op. Took water eagerly after op.	Some bleeding for 2 weeks. Severe hæmorrhage 7.45 a. m. 6/30/15.

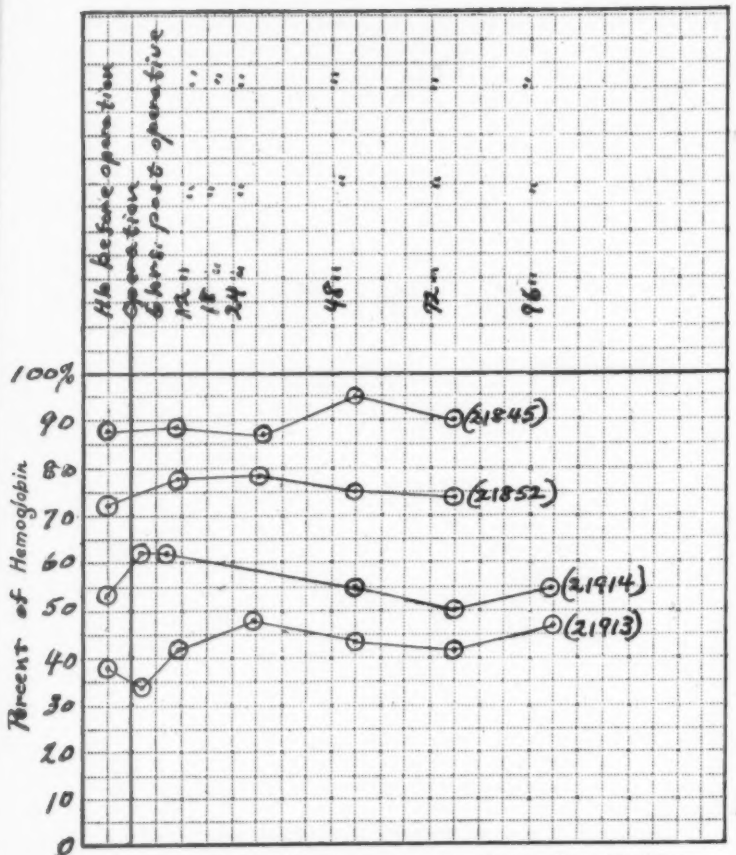

CHART I.—Hemoglobin curves in cases of very slight blood loss.

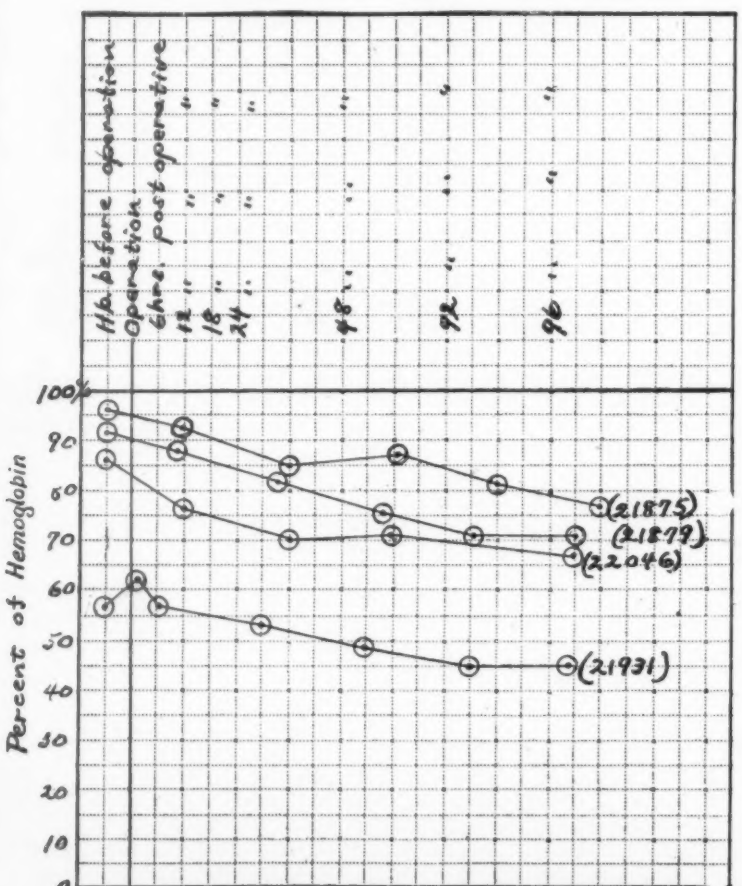

CHART II.—Hemoglobin curves in cases of moderate blood loss.

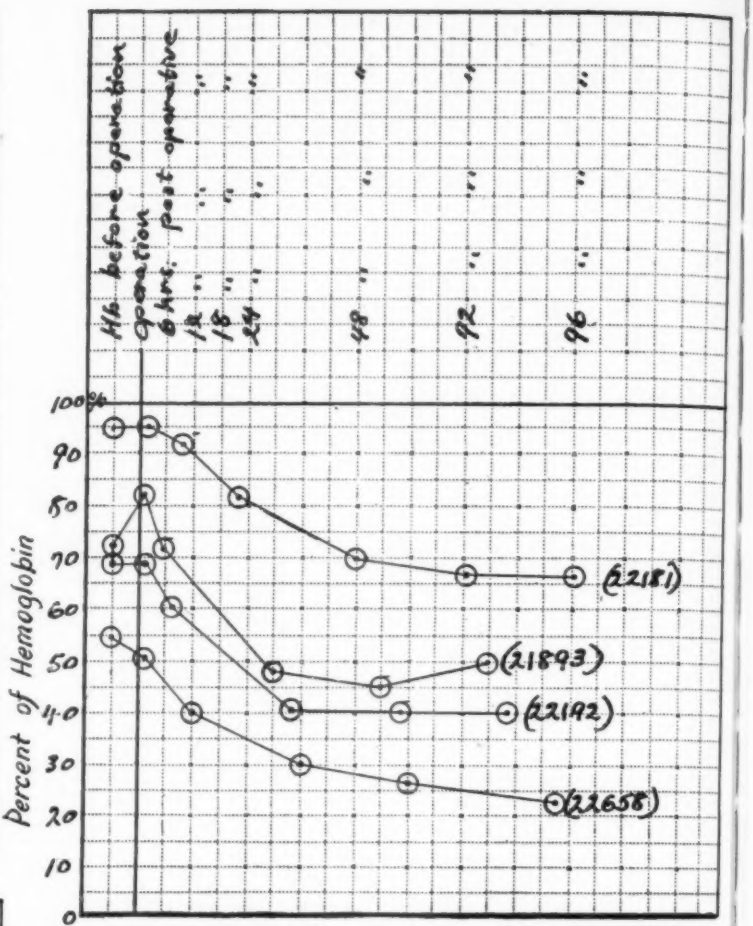

CHART III.—Hemoglobin curves in cases of severe hemorrhage.

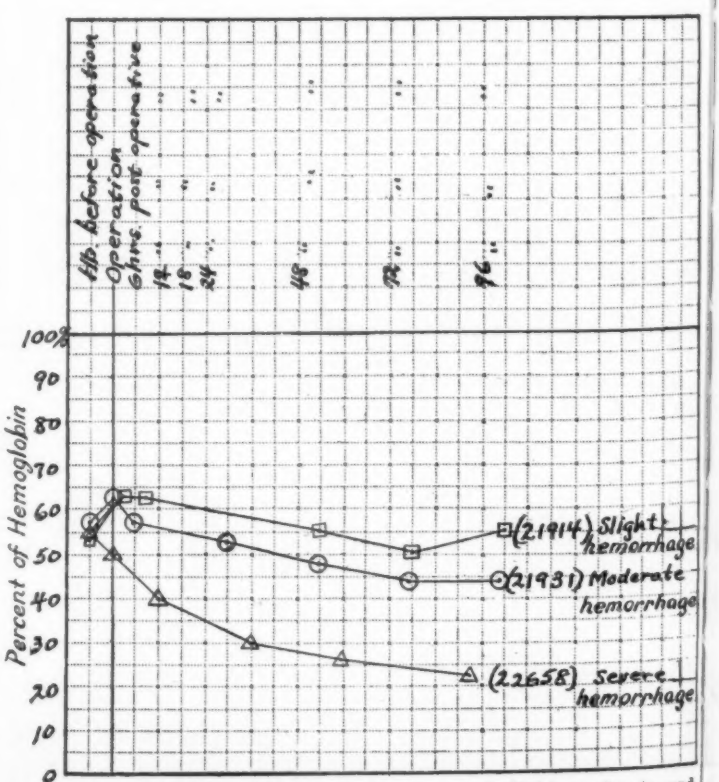

CHART IV.—Hemoglobin curves in cases of slight, moderate and severe hemorrhage.

MEMBERS OF THE JOHNS HOPKINS HOSPITAL AND DISPENSARY STAFF ENGAGED IN MILITARY DUTY

DR. WILLIAM H. WELCH, Major, M. O. R. C., special duty, Surgeon General's Office.

DR. WINFORD SMITH, Major, M. O. R. C., special duty, Surgeon General's Office.

DR. THEODORE C. JANEWAY, Major, M. O. R. C., special duty, Surgeon General's Office.

DR. WILLIAM S. THAYER, Major, M. O. R. C., Red Cross Commission to Russia.

DR. JOHN M. T. FINNEY, Major, M. O. R. C., Chief of Surgical Staff, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. THOMAS R. BOGGS, Major, M. O. R. C., Chief of Medical Staff, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. GEORGE WALKER, Major, M. O. R. C., Adjutant and Surgeon, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. HUGH H. YOUNG, Major, assigned to British medical service.

DR. THOMAS B. FUTCHER, Consultant, Canadian Military Hospital, B. E. F., Orpington, Kent, England.

DR. WILLIAM S. BAER, Captain, M. O. R. C., Assistant Surgeon, (Orthopedic) U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. F. H. BAETJER, Major, M. O. R. C., on active duty, Johns Hopkins Hospital.

DR. CLYDE G. GUTHRIE, Captain, M. O. R. C., Assistant Physician, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. GEORGE J. HEUER, Captain, M. O. R. C., Assistant Surgeon (Brain Surgery), U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. WALTER A. BAETJER, Captain, M. O. R. C., Chief of laboratory staff, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. CHARLES BAGLEY, Captain, M. O. R. C., assigned to duty in Surgeon General's Office.

DR. BERTRAM M. BERNHEIM, Captain, M. O. R. C., Assistant Surgeon (Vein Surgery), U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. EVELETH W. BRIDGMAN, Captain, M. O. R. C., Assistant Physician and Registrar, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. JOHN STAIGE DAVIS, Captain, M. O. R. C., on active duty, Baltimore.

DR. WILLIAM A. FISHER, Captain, M. O. R. C., Assistant Surgeon, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. HARVEY B. STONE, Captain, M. O. R. C., Quartermaster, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. STANHOPE BAYNE-JONES, Captain, M. O. R. C., U. S. Army Medical Relief Corps, France.

DR. DAVID M. DAVIS, 1st Lieutenant, M. O. R. C., on active duty in France.

DR. H. C. SCHMEISSER, 1st Lieutenant, M. O. R. C., Pathologist, Albany Base Hospital.

DR. J. ALBERT CHATARD, 1st Lieutenant, M. O. R. C., on active duty, Fort McHenry.

DR. J. A. C. COLSTON, 1st Lieutenant, M. O. R. C., on active duty, Medical Department, British Army in France.

DR. FRANK A. EVANS, 1st Lieutenant, M. O. R. C., on active duty, Medical Department, British Army in France.

DR. JOHN H. KING, 1st Lieutenant, M. O. R. C., Assistant Physician, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. J. H. M. KNOX, JR., Red Cross Commission to France.

DR. E. A. PARK, Red Cross Commission to France.

DR. JOHN T. KING, JR., 1st Lieutenant, M. O. R. C., on active duty, assigned to J. H. H.

DR. WILLIAM L. MILLEA, 1st Lieutenant, M. O. R. C., on active duty, Camp Oglethorpe, Ga.

DR. HENRY R. SLACK, 1st Lieutenant, M. O. R C., Assistant Surgeon (Laryngology), U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. HENRY L. SMITH, 1st Lieutenant, M. O. R. C., on active duty, Fort Oglethorpe, Ga.

DR. DANIEL D. V. STUART, Captain, M. O. R. C., on active duty, Washington, D. C.

DR. GEORGE L. STICKNEY, 1st Lieutenant, M. O. R. C., on active duty, Medical Department, British Army in France.

DR. ALAN C. SUTTON, 1st Lieutenant, M. O. R. C., on active duty, Medical Department, British Army in France.

DR. CHARLES A. WATERS, 1st Lieutenant, M. O. R. C., Roentgenologist, U. S. Army Base Hospital No. 18, American Expeditionary Force, France (Hopkins Base Hospital).

DR. CLARENCE A. NEYMANN, 1st Lieutenant, M. O. R. C., New York Psychiatric Unit.

Former members of the Hospital and Dispensary Staff who resigned during the past year to take up active military duty:

DR. EVERETT D. PLASS.
DR. DANIEL DAVIS.
DR. AUBREY T. MUSSEN.
DR. JOHN C. LYMAN.
DR. RAYMOND S. HUSSEY.
DR. VIRGIL P. W. SYDENSTRICKER.
DR. H. N. SHAW.
DR. L. R. WHARTON.
DR. CHARLES L. MCCARTHY.
DR. HOWARD E. ASHBURY.
DR. ERNEST S. DU BRAY.
DR. GEORGE R. DUNN.
DR. J. P. EDISON.
DR. R. W. HALL.
DR. W. D. JACK.
DR. U. R. MASON.
DR. T. L. SUTTON.
DR. C. E. SEVIER.
DR. D. G. SMITH.
DR. H. C. SCHMEISSER.
DR. D. C. W. SMITH.
DR. H. W. REID.
DR. J. E. MOORE.
DR. M. K. MILLER.
DR. P. F. MCGUIRE.
DR. L. K. MCCAFFERTY.
DR. W. B. MARTIN.
DR. I. K. LOVETT.
DR. J. A. ETHERIDGE.
DR. H. C. BEAN.
DR. LAWRENCE REYNOLDS.
DR. G. A. STEWART.
DR. ROADES FAYERWEATHER.
DR. L. C. SPENCER.
DR. ELIZABETH HURDON.
DR. H. L. CECIL.
DR. N. M. KEITH.
DR. G. H. PRESTON.
DR. N. WORTH BROWN.

NOTES AND NEWS

Dr. Dana W. Atchley is Instructor in Medicine and Instructor in Clinical Pathology, College of Physicians and Surgeons, Columbia University, New York City; and Assistant Physician, Presbyterian Hospital.

Dr. Frank C. Beall is Surgeon-in-Charge, the Johnson-Beall Hospital, Fort Worth, Texas.

Dr. Barney Brooks is Associate in Surgery, Washington University Medical School, and Visiting Surgeon, Barnes Hospital, St. Louis, Mo.

Dr. S. W. Budd is Associate Professor of Pathology and Associate in Medicine, Medical College of Virginia, Richmond, Va.

Dr. Walter C. Burket is First Lieutenant, Medical Reserve Corps. He is at present with Field Hospital No. 19, which is stationed at Fort Riley, Kansas.

Dr. Montrose T. Burrows is Associate Professor of Pathology and Acting Professor of Pathology, Washington University Medical School, St. Louis, Mo.

Dr. C. N. B. Camac is a Major in the Medical Reserve Corps, and Medical Chief of the Base Hospital, Fort McPherson, Ga. He is also Instructor in the School of Gas Defense, United States Army.

Dr. John R. Caulk is Associate in Clinical Genito-Urinary Surgery, Washington University; Assistant Surgeon to Barnes Hospital; Chief of Clinic, Genito-Urinary Department, Washington University Dispensary; Genito-Urinary Surgeon to St. Luke's Hospital, St. Louis, Mo.

Dr. John W. Churchman is Professor of Surgery, Yale University; Visiting Surgeon, New Haven Hospital; Acting Head of the Department of Surgery; Acting Chief Surgeon, New Haven Hospital and New Haven Dispensary.

Dr. S. W. Clausen is Resident Physician, Children's Hospital, St. Louis, Mo.

Dr. Henry Wireman Cook is Assistant in Medicine, the University of Minnesota.

Dr. C. D. Cowles, Jr., is Major in the Medical Corps, U. S. A., and is stationaed at Fort Oglethorpe, Ga.

Dr. Arthur W. Elting is Major in the Medical Reserve Corps and Director of Base Hospital No. 33.

Dr. Clarence B. Farrar is Captain in the Canadian Army Medical Corps and is connected with the Psychiatric Military Hospital, Ottawa, Canada.

Dr. L. W. Gorham is Instructor in Medicine, Albany Medical College, and Assistant Attending Physician, Albany Hospital. He is a member of the Medical Reserve Corps and is Chief of Medical Service, Base Hospital No. 33.

Dr. R. L. Haden is Director of Laboratories, Henry Ford Hospital, Detroit, Mich.

Dr. C. W. Hennington is Major in the Medical Reserve Corps, and Assistant Director and Chief of the Surgical Service, Base Hospital No. 19.

Dr. James M. Hitzrot is Assistant Professor of Clinical Surgery, Cornell University Medical College.

Dr. August Hoch is editor of the Psychiatric Bulletin. Address: Montecito, Cal.

Dr. Samuel H. Hurwitz is Assistant Clinical Professor of Medicine, Medical Department, University of California.

Dr. Clarence B. Ingraham is Professor of Gynecology and Obstetrics, the University of Colorado. He is Captain in the Medical Reserve Corps and is stationed at present at Fort Riley, Kansas.

Dr. Theodore C. Janeway is a member of the General Medical Board of Council of National Defense.

Dr. Harry L. Langnecker is Passed Assistant Surgeon, U. S. N. R. F. He is Orthopedist to the Naval Base Hospital Unit No. 2.

Dr. D. Sclater Lewis is with No. 3 Canadian General Hospital, B. E. F., France.

Dr. Irving P. Lyon is Assistant Professor of Medicine, University of Buffalo, and Attending Physician, Buffalo General Hospital.

Dr. W. G. MacCallum is Professor of Pathology and Bacteriology, Johns Hopkins University, and Pathologist to the Johns Hopkins Hospital.

Dr. R. H. Major is Professor of Pathology, University of Kansas, Rosedale, Kansas.

Dr. Kenneth F. Maxcy is a member of the Medical Staff, Henry Ford Hospital, Detroit, Mich.

Dr. William B. McClure is Fellow in the Otho A. Sprague Memorial Institute, Chicago, Ill.

Dr. Carl R. Meloy is Director of Laboratories, the Grace Hospital, Detroit, Mich.

Dr. George R. Minot is Assistant in Medicine, Harvard Medical School, Assistant in Medicine, Massachusetts General Hospital, and holder of the Dalton Research Fellowship, Massachusetts General Hospital.

Dr. Charles F. Nassau is Assistant Professor of Surgery, Jefferson Medical College; Chief Surgeon, Frankford Hospital; Surgeon, St. Joseph's Hospital; Assistant Surgeon, Jefferson Medical College Hospital; and Consulting Surgeon, Pottstown Hospital, Pottstown, Pa.

Dr. C. D. Parfitt is Special Adviser in Tuberculosis, Toronto General Hospital.

Dr. H. W. Plaggemeyer is Instructor in Surgery, Detroit Medical College, Chief of Staff, Department of Urological Surgery, Grace Hospital, and Junior Attending Surgeon, Harper Hospital, Detroit, Mich.

Dr. J. P. Pratt is Assistant Surgeon, Henry Ford Hospital, Detroit, Mich.

Dr. D. Maxwell Ross is in charge of mental wards, 52d General Hospital, B. E. M. F., Salonika.

Dr. Peyton Rous is Associate Member, Rockefeller Institute for Medical Research, New York City.

Dr. Lewis A. Sexton is Superintendent, the Hartford Hospital, Hartford, Conn.

Dr. W. F. Shallenberger is Associate Professor of Gynecology, Emory University (Atlanta Medical College); Visiting Gynecologist, Georgia Baptist Hospital, and Assistant Visiting Gynecologist, Grady Hospital, Atlanta, Ga.

Dr. William Sharpe is Attending Neurologist to the Beth Israel Hospital, New York City.

Dr. Winford H. Smith is a member of the General Medical Board of Council of National Defense.

Dr. R. R. Snowden is a First Lieutenant in the Medical Reserve Corps. He is on duty in France with Base Hospital Unit No. 27.

Dr. A. R. Stevens is a member of the Medical Reserve Corps and is with the N. Y. Presbyterian Base Hospital Unit in France.

Dr. Solomon Strouse is Assistant Professor of Medicine, Northwestern University School of Medicine.

Dr. Martin B. Tinker is a Major in the Medical Reserve Corps. He is stationed at present at Fort Riley, Kansas.

Dr. Donald V. Trueblood is a First Lieutenant in the Medical Reserve Corps, and has been assigned to duty with the American Expeditionary Force in France.

Dr. P. S. Tucker is First Lieutenant, Medical Corps, U. S. A., and is stationed at Fortress Monroe, Va.

Dr. J. H. J. Upham is Professor of Medicine and Acting Head of the Department, Ohio State University School of Medicine, and President of the Ohio State Medical Board.

Dr. K. H. Van Norman is Assistant to Assistant Director Medical Services (A. D. M. S.) Military District No. 2; headquarters, Toronto.

Dr. L. M. Warfield is Assistant Superintendent, Milwaukee County Hospital, Wauwatoso, Wis., and Professor of Clinical Medicine at Marquette Medical School, Milwaukee, Wis.

Dr. Ernest M. Watson is Assistant Visiting Urologist to the Municipal Hospital and to the Erie County Hospital, Buffalo, N. Y.

Dr. Charles H. Watt is Surgical Director of the Union Hospital, Fall River, Mass.

Dr. Jerome P. Webster is First Lieutenant Medical Reserve Corps, and is stationed at Fort Benjamin Harrison, Ind.

Dr. William H. Welch is Director of the School of Hygiene to be established in connection with the Johns Hopkins University.

Dr. Milton C. Winternitz is Professor of Bacteriology and Pathology, Yale University School of Medicine, and Pathologist to the New Haven Hospital.

Dr. Paul G. Woolley is Mary M. Emery Professor of Pathology, University of Cincinnati. He is a Captain in the Medical Reserve Corps, and is stationed at Fort Oglethorpe, Ga.

Dr. J. Kent Worthington is Assistant in Urology, Indiana University School of Medicine; Assistant and Attending Urologist, City Hospital; and Attending Urologist, City and Babbs Dispensary. He is a Captain in the Medical Reserve Corps, and is stationed at present at Fort Benjamin Harrison, Ind.

Dr. Charles B. Wright is Chief of the Gastro-Intestinal Clinic, University Dispensary, and Assistant Visiting Physician, University Hospital, Minneapolis, Minn.

BOOKS RECEIVED

Diseases of the Genito-Urinary Organs and the Kidney. By Robert Holmes Greene, A. M., M. D. and Harlow Brooks, M. D. Fourth edition, thoroughly revised. 1917. 8°. 666 pages. W. B. Saunders Company, Philadelphia and London.

The Treatment of Tabetic Ataxia by Means of Systematic Exercise. By Dr. H. S. Frenkel. Second revised and enlarged English edition by L. Freyberger, J. P., M. D. (Vienna), M. R. C. P. Lond., M. R. C. S. Eng. With 130 illustrations. 1917. 8°. 209 pages. P. Blakiston's Son & Co., Philadelphia.

Dr. Lyman Spalding, the Originator of the United States Pharmacopœia. Co-laborer with Dr. Nathan Smith in the Founding of the Dartmouth Medical School and its First Chemical Lecturer; President and Professor of Anatomy and Surgery of the College of Physicians and Surgeons of the Western District, at Fairfield, N. Y. By his grandson, Dr. James Alfred Spalding. 1916. 380 pages. W. M. Leonard, Boston.

Operative Surgery of the Nose, Throat, and Ear. For Laryngologists, Rhinologists, Otologists, and Surgeons. By Hanau W. Loeb, A. M., M. D. in collaboration with Joseph C. Beck, M. D., George W. Crile, M. D., William H. Haskin, M. D., Robert Levy, M. D., Harris P. Mosher, M. D., George L. Richards, M. D., George E. Shambaugh, M. D., and George B. Wood, M. D. In two volumes. Volume II. Four hundred and seventy-six illustrations. 1917. 8°. 427 pages. C. V. Mosby Company, St. Louis.

Asthma, Presenting an Exposition of the Nonpassive Expiration Theory. By Orville Harry Brown, A. B., M. D., Ph. D., with a foreword by George Dock, Sc. D., M. D. Thirty-six engravings. 1917. 8°. 330 pages. C. V. Mosby Company, St. Louis.

Experimental Pharmacology. By Dennis E. Jackson, Ph. D., M. D. With three hundred and ninety original illustrations including twenty-four full-page color plates. 1917. 8°. 536 pages. C. V. Mosby Company, St. Louis.

Impotency, Sterility and Artificial Impregnation. By Frank P. Davis, Ph. B., M. D. 1917. 12°. 138 pages. C. V. Mosby Company, St. Louis.

The Climate and Weather of San Diego, California. Prepared under the direction of Willis L. Moore, Chief United States Weather Bureau. By Ford A. Carpenter, Local Forecaster. Illustrated with photographs and charts by the author and others. 1913. 16°. 118 pages. Published by the San Diego Chamber of Commerce.

The Adventure of Death. By Robert W. Mackenna. 1917. 12°. 197 pages. G. P. Putnam's Sons, New York and London.

Acute Poliomyelitis. By George Draper, M. D., with a foreword by Simon Flexner. With 19 illustrations. 1917. 8°. 149 pages. P. Blakiston's Son & Co., Philadelphia.

Psychological Medicine. A Manual on Mental Diseases. For Practitioners and Students. By Maurice Craig. Third edition. With 27 plates, some in colour. 1917. 8°. 484 pages. P. Blakiston's Son & Co., Philadelphia.

Chemical and Microscopical Diagnosis. By Francis Carter Wood, M. D. Third edition. With 194 illustrations in the text and 10 plates, nine of which are colored. 1917. 8°. 791 pages. D. Appleton & Company, New York and London.

U. S. Department of Commerce, Bureau of the Census. Mortality Statistics, 1915. Sixteenth Annual Report. Sam L. Rogers, Director. 1917. 707 pages. Government Printing Office, Washington.

Saint Thomas's Hospital Reports. New Series. Edited by Dr. J. J. Perkins and Mr. C. A. Ballance. Volume XLIII. 1914. 8°. 303 pages. 1916. J. & A. Churchill, London.

Some Personal Recollections of Dr. Janeway. By James Bayard Clark. 1917. 12°. 36 pages. G. P. Putnam's Sons, New York and London.

American Association for Study and Prevention of Infant Mortality. Transactions of the Seventh Annual Meeting, Milwaukee, October 19-21, 1916. 1917. 8°. 364 pages. Press of Franklin Printing Company, Baltimore.

Notes on Galvanism and Faradism. By E. M. Magill, M. B., B. S. Lond., D. P. H., R. C. S. I. (Hons.) With 67 illustrations. 1916. 12°. 220 pages. Paul B. Hoeber, New York.

Urology. Diseases of the Urinary Organs, Diseases of the Male Genital Organs, the Venereal Diseases. By Edward L. Keyes, Jr., M. D., Ph. D. With 204 illustrations in the text and 18 plates, four of which are colored. 1917. 8°. 908 pages. D. Appleton & Company, New York and London.

Glaucoma. A Handbook for the General Practitioner. By Robert Henry Elliot, M. D., B. S. Lond., Sc. D. Edin., F. R. C. S. Eng., etc. Lieut.-Colonel I. M. S. (Retired). 1917. 8°. 60 pages. Paul B. Hoeber, New York.

The British Guiana Medical Annual. Twenty-first year of issue for 1915. Edited by A. J. Craigen, M. B., Ch. B. (Aberdeen) and F. G. Rose, B. A., M. B., B. C. (Cantab.) M. R. C. S., L. R. C. P. 1916. 8°. 179 pages. Printed by "The Argosy" Company, Limited, Demerara.

The Medical Association of the Isthmian Canal Zone. Proceedings for the half year, January to June, 1916. Volume IX, part 1. 1917. 8°. 190 pages. The Health Department, Panama Canal Zone.

Baltimore, Sub-Department of Health. Department of Public Safety. Annual Report to the Mayor and City Council of Baltimore for the Fiscal Year Ended December 31, 1915. 1916. 8°. 348 pages. Meyer & Thalheimer, Baltimore.

International Clinics. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles. By leading members of the medical profession throughout the world. Edited by H. R. M. Landis, M. D. Volume II. Twenty-seventh series. 1917. 8°. 313 pages. J. B. Lippincott Company, Philadelphia and London.

Collected Papers of the Mayo Clinic, Rochester, Minnesota. Edited by Mrs. M. H. Mellish, Vol. VIII, 1916. 8°. 1014 pages. 1917 W. B. Saunders Company, Philadelphia and London.

The Treatment of Emergencies. By Hubley R. Owen, M. D. With 249 illustrations. 1917. 12°. 350 pages. W. B. Saunders Company, Philadelphia and London.

Finch and Baines. A Seventeenth Century Friendship. By Archibald Malloch, B. A. (Queen's); M. D. (McGill); Temporary Captain, Canadian Army Medical Corps. 1917. 4°. 89 pages University Press, Cambridge.

Physical Exercises for Invalids and Convalescents. By Edward H. Ochsner, B. S., M. D., F. A. C. S. Illustrated. 1917. 12°. 54 pages. C. V. Mosby Company, St. Louis.

Roentgen Technic (Diagnostic.) By Norman C. Prince, M. D. With 71 original illustrations. 1917. 8°. 140 pages. C. V. Mosby Company, St. Louis.

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